“DERMOSCOPIC PATTERNS IN LICHENOID DERMATOSES”

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DISSERTATION SUBMITTED TO

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR, KARNATAKA,

IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE DEGREE OF

DOCTOR OF MEDICINE (M.D.)

IN

DERMATOLOGY, VENEREOLOGY AND LEPROSY

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APRIL 2019

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Department	DERMATOLOGY, VENEREOLOGY AND LEPROSY
Acceptable Maximum Limit	10%
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ACKNOWLEDGEMENT

One of the joys of completion of this dissertation is to look over the journey past and remember and thank all the people who have helped and supported me along this long but fulfilling road. First and foremost, I thank the Almighty for giving me the strength and ability to carry out this study.

I am deeply indebted and grateful to my guide, ***Dr. Rajashekar. T.S.*** *Professor and Head, Department of Dermatology, Venereology and Leprosy, Sri Devaraj Urs Medical College, for his able guidance, support, timely advice and constant encouragement throughout the period of the study.*

I thank ***Dr. Suresh Kumar K****, Assistant Professor, Department of Dermatology, Venereology and Leprosy, Sri Devaraj Urs Medical College, for his support and guidance during my post graduation.*

I would also like to warmly extend my gratitude to ***Dr. Sharanya Padma****, Senior Resident, Department of Dermatology, Venereology and Leprosy, Sri Devaraj Urs Medical College, for her constant encouragement.*

No words can express the gratitude I feel towards my beloved parents, ***Mr. B. C. Prem Kumar*** *and* ***Mrs. Mamatha Prem****, whose countless sacrifices and endless love has made me who I am today in life.*

*I also thank my sister **Mrs. Preethi Prem** for her motivation and for being a constant source of strength.*

*I am thankful to my postgraduate colleague, **Dr. Shruthi Madhavi. G**, my seniors and dear juniors for all their love, motivation and help.*

I am truly blessed in having the most wonderful friends and would like to thank them for their endless support.

I will be failing my duty if I do not thank all my patients involved in this study, without whose co-operation and patience this study would have been impossible.

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LIST OF ABBREVIATIONS USED

➢ LP	Lichen Planus
➢ WS	Wickham's striae
➢ LPH	Lichen Planus Hypertrophicus
➢ LPPe	Lichen planus pemphigoides
➢ LDE	Lichenoid drug eruption
➢ LPP	Lichen Planus Pigmentosus
➢ LPp	Lichen Planopilaris
➢ LS	Lichen Striatus
➢ DEJ	Dermoepidermal Junction
➢ BMZ	Basement Membrane Zone
➢ DIF	Direct immunofluorescence
➢ IIF	Indirect immunofluorescence

ABSTRACT

"DERMOSCOPIC PATTERNS IN LICHENOID DERMATOSES"

BACKGROUND:

Lichenoid dermatoses is a term used to describe various dermatological conditions which show interface dermatitis, on histopathology. Routinely, histopathological examination is done to diagnose these skin conditions but of late dermoscopy is being used in their diagnosis.

Dermoscopy is a non-invasive diagnostic methodology with the use of a specialized hand held device called dermoscope. It allows rapid and magnified observation of the skin and helps in visualization of the morphological features which are otherwise not visible to the naked eye.

Specific pigmentary and vascular patterns are observed with certain dermatoses and these could be used for their diagnosis. Dermoscopy is commonly used in evaluating pigmented lesions, but lately it is also being explored in various other skin conditions like lichenoid dermatoses, psoriasis, infections and infestations.

Dermoscopy may be used as an easy alternative to histopathological examination, especially in paediatric patients and over cosmetically important areas like face. Clinical use of dermoscopy in inflammatory dermatoses improves the diagnostic ability and the fundamental aspects in daily practice.

Though dermoscopy is an easy and useful diagnostic tool in inflammatory skin conditions, there is paucity of literature on dermoscopic findings in these dermatoses. This study will appraise our knowledge on the dermoscopic patterns in lichenoid dermatoses.

OBJECTIVES:

To perform dermoscopy and document the findings in lichenoid dermatoses

MATERIALS AND METHODS:

The present study was an observational study carried out in the Department of Dermatology at R.L Jalappa Hospital attached to Sri Devaraj Urs Medical College, Tamaka, Kolar from January 2017 to July 2018. One hundred patients who presented with lichenoid dermatoses and who satisfied the inclusion criteria were included in this study. A detailed history of the patient including name, age, sex, history of presenting illness, systemic disease, family history of similar complaints and drug intake were taken. A written informed consent was taken from the patients and in children from parents or guardian. General physical examination, cutaneous, mucosal and nail examination were done in all cases. The cutaneous lesions were examined directly through the dermoscope Dermlite DL3N with 25mm 10x magnification using both polarized and non-polarized mode. Then, the dermoscopic photographs were

taken with the help of an adaptor attached to an iphone 8 camera with a 12x zoom.

RESULTS:

Among the 100 cases, 51 were female and 49 were male. The youngest subject in the study was 0.9 year old and the oldest subject was 84 years old. The mean age of the study group was 29.34+/- 19.48. The maximum number of cases 23% were seen in age group of 0-10 years, and the minimum of cases 1% were noted the age group of more than 80 years. Among the 100 cases, Lichen Planus was 45% and its variants i.e Lichen planus hypertrophicus was 5%, Actinic Lichen planus was 2%. Other lichenoid dermatoses screened in the present study were Lichenoid drug eruption (1%), Lichen planus pigmentosus (5%), Lichen planopilaris (7%), Lichen nitidus (27%), Lichen striatus (8%). Patients with Lichen planus (71.1%) and Lichen nitidus (74.1%) gave 0-3 months history of duration of lesions whereas patients with Lichen planus pigmentosus (40%) and Lichen planopilaris (42.8%) gave more than 12 months history of duration of illness. History of itching was seen in all cases of Lichen Planus, Lichen planus hypertrophicus and Actinic Lichen planus whereas asymptomatic lesions were seen in all cases of Lichen planus pigmentosus, Lichen nitidus and Lichen striatus. Loss of hair was seen in all cases of Lichen planopilaris. On examination, papules were commonly seen in Lichen Planus

(64.4%), Lichen nitidus (100%) and Lichen striatus (100%). Macules were seen in Lichen planus pigmentosus (100%) and alopecia in Lichen planopilaris (85.7%). The most common site of involvement was lower limbs in Lichen Planus (88.8%) and Lichen planus hypertrophicus (100%) followed by upper limbs in Lichen nitidus and Lichen striatus. In Lichen planopilaris (85.7%) cases involved scalp and in Lichen planus pigmentosus face (100%) and upper limb (60%) was commonly involved. Oral and genital mucosa was involved in 64.4% and 11.1% cases of Lichen planus respectively and nail involvement with Lichen planus was seen in 26.6%. WS was seen only in Lichen planus in 91.1% cases. The common morphological pattern of WS seen was radial streaming pattern (63%) and reticulate pattern (41.4%) with white colour WS (82.9%) being commonest. Brown globules was the commonest pigment pattern seen in Lichen planus (60%), Lichen planus hypertrophicus (80%) and Lichen planus pigmentosus (100%). Greyish blue globules seen in Lichen planopilaris (71.4%), Lichen planus hypertrophicus (100%), Lichen planus (17.7%), Lichen planus pigmentosus (20%). Pigment pattern was absent in Lichen nitidus (100%) and Lichen striatus (100%). Diffuse pattern of pigmentation was seen in Lichen planus (100%), Lichen planus hypertrophicus (100%) and Lichen planus pigmentosus (100%). Perifollicular type of pigmentation was seen in Lichen planus pigmentosus (100%) and Lichen planopilaris (71.4%). Red globules (40%) and peripheral homogenous erythema (6.6%) was seen in Lichen planus. No vascular patterns were seen in Lichen Planus (46.6%), Lichen planus

hypertrophicus (80%), Lichen planus pigmentosus (100%), Lichen planopilaris (85.7%), Lichen nitidus (100%) and Lichen striatus (100%). White globules were seen in Lichen nitidus (100%) and coalescing white globules were seen in lichen striatus (100%). In Lichen planopilaris, empty follicles (100%), perifollicular scales and casts (100%) and structureless areas (57.1%) were seen.

CONCLUSION:

- Dermoscopy helps in identifying fine structures which are not visible to the naked eye.
- Wickham's striae is the most common dermoscopic finding in Lichen Planus and other features like melanophages and blood vessels can also be visualized which helps in identifying the disease activity.
- Clinical use of dermoscopy in lichenoid dermatoses improves the diagnostic ability, reduces the need for biopsy and to know the prognosis of the disease.

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INTRODUCTION

INTRODUCTION

Lichenoid dermatoses is a term used to describe various dermatological conditions which show interface dermatitis, on histopathology. The lichenoid dermatoses are Lichen planus and its variants, Lichenoid drug eruption, Lichen striatus, Lichen nitidus, Benign lichenoid keratosis, Ashy dermatosis, Graft versus host disease, Dermatomyositis and Lichenoid reaction in seborrheic keratosis (irritant type).[1]

Dermoscopy is a non-invasive diagnostic methodology which allows rapid and magnified observation of the skin and helps in visualization of the morphological features which are otherwise not visible to the naked eye.[2]

Dermoscopy may be used as an easy alternative to histopathological examination, especially in paediatric patients and over cosmetically important areas like face.[3] Clinical use of dermoscopy in inflammatory dermatoses improves the diagnostic ability and the fundamental aspects in daily practice.[4]

Though dermoscopy is an easy and useful diagnostic tool in inflammatory skin conditions, there is paucity of literature on dermoscopic findings in these dermatoses.[4] This study will appraise our knowledge on the dermoscopic patterns in lichenoid dermatoses.

AIMS AND OBJECTIVES

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To perform dermoscopy and document the findings in lichenoid dermatoses

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Lichenoid dermatoses are a heterogeneous group of diseases with varied clinical presentations and histologically by a band like lymphocytic infiltrate distributed in the papillary dermis.[5] The lichenoid dermatoses are Lichen planus (LP) and its variants, Lichenoid drug eruptions, Lichen striatus, Lichen nitidus, Benign lichenoid keratosis, Ashy dermatosis, Graft versus host disease, Dermatomyositis and Lichenoid reaction in seborrheic keratosis (irritant type).[6] LP is the prototype.[1]

HISTORICAL ASPECTS

LP was first described by Von Hebra, which he named as "leichen ruber." The term "lichen planus" was coined by Erasmus Wilson in 1869. The word "leichen" from greek means "tree moss" and the word "planus" from Latin means "flat". The "Wickham's striae" was first described by Wickham in 1895.[1]

HISTORY OF DERMOSCOPE

Various terms were used to describe dermoscopy, namely, dermatoscopy, surface microscopy, incident light microscopy, and epiluminescence light microscopy.[7]

Skin surface microscopy was started in 1663 with Kolhaus who investigated the small vessels in the nailfold with the help of a microscope.[8]

In 1878, oil immersion in light microscopy was described by Abbe and the similar principle was used in skin surface microscopy by the German dermatologist, Unna, in 1893. He introduced the term ‘‘diascopy’’ and described the use of immersion oil and a glass spatula in lichen planus and lupus erythematosus.[8]

The term ‘‘dermatoscopy’’ was introduced by Johann Saphier, a German dermatologist in 1920. He published a series of communications using a new tool for skin examination which resembled a binocular microscope with a built-in light source.[9]

In the 1950s, Goldman in United States who further modified skin surface microscopy, published a series of interesting articles on a new device called ‘‘Dermoscopy’ and was the pioneer in the use of this technique for evaluation of pigmented skin lesions.[8]

In 1971, Rona MacKie was the first to identify the advantage of surface microscopy for the better preoperative diagnosis of pigmented skin lesions and for the differential diagnosis of benign versus malignant lesions.[8]

In 1981, Fritsch P and Pechlaner R differentiated benign and malignant skin lesions on the basis of various pigment network features. Pehamberger H et al introduced pattern analysis for the diagnosis of pigmented skin lesions in 1987.[10]

Further investigations were continued mainly in Europe by several Austrian and German groups.[9]

Large dedicated dermoscopic devices were first used in the late 1980s and hand‐held devices started developing in the early 1990s.[7]

The first Consensus Conference on Skin Surface Microscopy was held in 1989 in Hamburg and the Consensus Net meeting on Dermoscopy, which was held in 2001 in Rome, was the first international meeting of its kind.[8]

DERMOSCOPE

Dermoscopy is a non-invasive diagnostic technique, which aids in visualising very fine patterns of skin lesions and subsurface skin structures which are not visible to the naked eye.[11]

A dermoscope works similar to a magnifying lens, with the added features of an inbuilt illuminating system and with higher magnification.[12] The

'sub-macroscopic' view of lesions enhances clinical assessment. The advantage of dermoscope is the ability to assess structures as deep as in the reticular dermis and the ability to record images. It forms a link between macroscopic clinical dermatology and microscopic dermatopathology. The 'sub-macroscopic' view of lesions enhances clinical assessment.[13]

PRINCIPLE

Dermoscopy works on the principal of "transillumination" of the lesion.[12] Light incident on skin undergoes reflection, refraction, diffraction and absorption. The phenomenon of light reflecting back after an incident on the skin surface is called glare or specular reflectance. Light gets reflected back because of higher refractive index (1.55) of the stratum corneum, when compared to air (1.0). Thus, most of the light incident on the skin surface is reflected when visualized with a magnifying glass.[14]

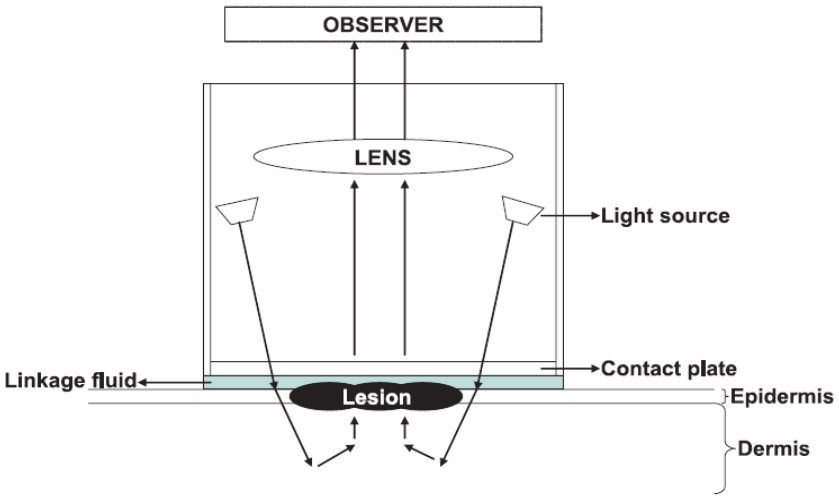

Figure 1: Optics of dermoscope. The refracted light transilluminatesthe lesion while passing through it and is perceived as a distinct pattern.[12]

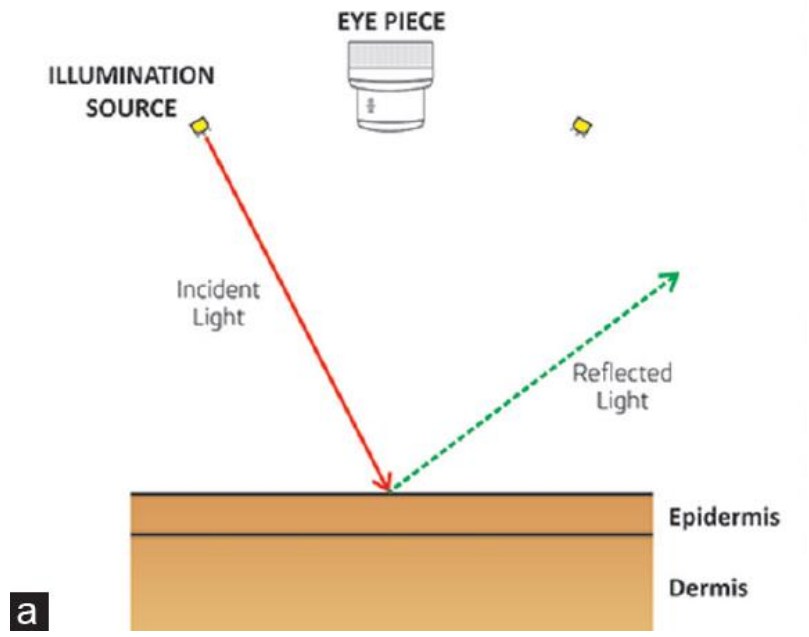

Figure 2: Magnified light, Incident light is reflected back from skin surface.[14]

Specular reflectance reduction is achieved by a technology called polarized dermatoscopy. Here, two filters are held orthogonally at 90°. Polarized light which incidents on the stratum corneum gets partly reflected from the surface and the rest of the light enters the skin. The part which gets reflected from the skin surface will maintain its polarization and gets blocked by the second filter. The part of light which enters the surface of skin will lose its polarization and hence it is allowed to pass the second filter. The polarized light penetrates 60–100 mm deep into the skin surface. (Figure 4).[14]

The technology of allowing the light which has lost its polarization to pass through the second filter whereas blocking the light that maintains polarization is known as cross polarization.[14]

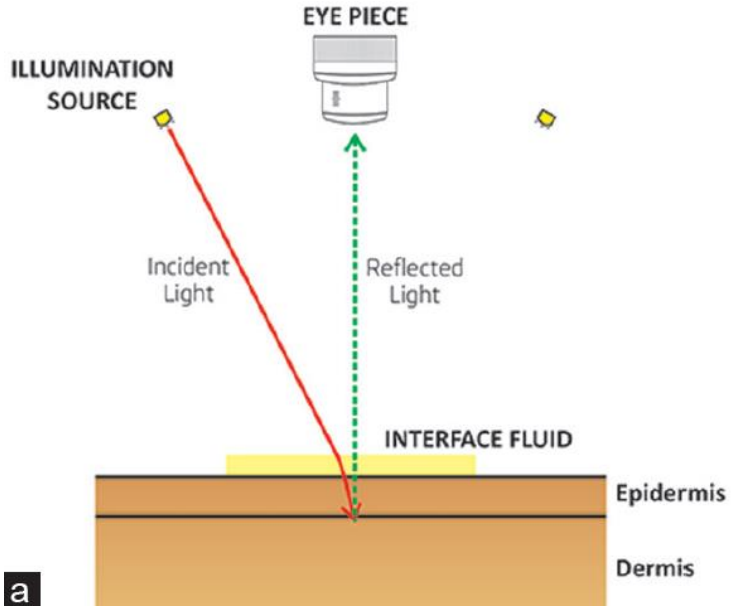

Figure 3: Nonpolarized dermatoscopy. Incident light in the presence of interface fluid penetrates the superficial layers of the skin.[14]

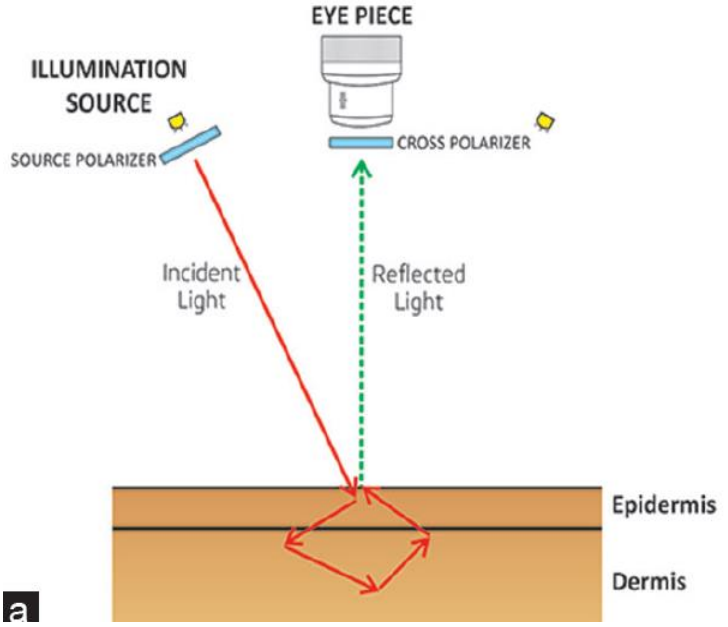

Figure 4: Polarized dermatoscopy. Incident polarized light penetrates deeper, loses polarization and is allowed to the eyepiece by the cross-polarizer.[14]

BASIC DESIGN OF A DERMOSCOPE

The essential components of a dermoscope are:

1. Achromatic lens: Most of the instruments provide 10x magnification, higher magnifications if required can be achieved with special lenses.
2. Inbuilt illuminating system: Halogen lamps are placed within the handheld piece which are oriented at an angle of 20 degree. The colour contrasts of lesions are altered by the yellow light of halogen lamp. Light emitting diodes (LED) provide high intensity white light and consume 70% less power than halogen lamps.
3. Power supply: Handheld instruments are usually powered by batteries like lithium ion, AA battery, rechargeable lithium battery and rechargeable handles.
4. Contact plate- These are multicoated silicone glass and either graduated or nongraduated. Graduated plates have a scale inscribed to measure the dimensions of the lesion examined.[12]

TYPES OF DERMOSCOPY INSTRUMENTS

For simplicity, dermoscopic instruments can be grouped as:

a) Instruments without image capturing facility e.g. Dermoscope (Figure 5)

b) Instruments with image capturing facility e.g. Dermaphot (Figure 6)

c) Instruments with image capturing facility and analytical capability e.g. DermoGenius Molemap[12] (Figure 7)

Figure 5: **Dermoscope with image capturing capability. Taking dermoscopic images by placing the extended camera lens over the eyepiece.**[7]

Figure 6: Dermaphot, a dermoscope with image capturing capability.[12]

Figure 7: DELTA 20, a dermoscope with camera attachable facility. A, rechargeable handle; B, hand-piece; C, connector ring; D, photo-adaptor; E, camera; F, contact plate; G, small contact plate.[12]

TECHNIQUE

Dermoscopy can be done by both contact and non-contact technique. In the contact technique, the glass plate of the instrument and the surface of the linkage fluid applied on the lesion are in contact whereas there is no contact of the lens with the skin in non-contact technique.[12]

Most of the light incident on dry, scaly skin is reflected, but smooth, oily skin allows most of the light to penetrate reaching the deeper dermis. This

principle is being used to improve the visibility of skin subsurface structures using linkage fluid over the lesions as it improves the translucency of the skin.[14]

The refractive index of the fluid interface should ideally be equal to that of the skin to match it optically as it minimizes the glare, allowing more light to penetrate through the stratum corneum.[13]

Various linkage fluids used are oils (immersion oil, mineral oil and olive oil) water, glycerine, antiseptic solution, alcoholic disinfectant, aqueous disinfectant, 70% ethanol, 90% isopropanol, liquid paraffin, water, and ultrasound gel.[14] Although the surface under ultrasound gel appears blurry, it is the fluid of choice in nail, mucosa, and periorbital region where other fluids will flow off.[13] Immersion oil is not used because it contains chlorinated paraffin and dibutyl phthalate which have teratogenic, fetotoxic, and carcinogenic effects. Water or antiseptic solutions are less preferred than oils as they evaporate quickly. Liquid paraffin which is safe, easily available, inexpensive provides good visibility. Glass has a refractive index (1.52) similar to that of skin (1.55) and hence when placed over oil-applied skin, further enhances transillumination of the lesion.[14]

The advantage of non-contact technique is no nosocomial infections but has a disadvantages of decreased illumination and poor resolution.[14]

Polarized light dermoscopy also avoids the possibility of cross infection from the contact plate when visualizing infectious dermatoses. This can be

avoided by use of disposable contact caps, adhesive tapes, food wraps and using 70% ethanol as interface medium.[14]

Polarized dermoscope allows better recognition of deeper structures (dermal pigment deposit, vasculature, fibrosis) than superficial layers of epidermis.[15]

The small contact area of the plate facilitate its use in difficult to access areas like the flexures, web spaces and nail fold capillaroscopy.[12]

Contact plates can be sterilized by using 2% glutaraldehyde, methylated spirit, boiling or autoclaving.[12]

APPLICATIONS

- Classic dermoscopy: In diagnosis of pigmented and non-pigmented skin tumors including melanocytic and non-melanocytic benign and malignant skin tumors.
- Entomodermoscopy: In diagnosis of skin infections and infestations caused by parasites, viral, bacterial, fungal or protozoan infections.
- Inflammoscopy: In diagnosis of skin diseases such as psoriasis, lichen planus, pityriasis rosea.
- Granulomatous skin diseases- sarcoidosis, lupus vulgaris, cutaneous leishmaniasis, granulomatous rosacea.

- Autoimmune diseases: Discoid lupus erythematosus, Lichen sclerosus and morphoea.
- Trichoscopy: for diagnosing hair and scalp disorders.
- Capillaroscopy: of the nail fold capillaries for the screening of autoimmune diseases.
- Dermoscopy for treatment decision and monitoring.[16]

DERMOSCOPIC FINDINGS

Dermoscopy is performed in inflammatory and infectious conditions where few parameters have to be assessed. They are (i) morphological vascular patterns (ii) arrangement of vascular structures (iii) colours and (iv) follicular abnormalities and other specific features.[16]

Some dermoscopic criteria appear to be highly specific for a particular condition, while others can be seen in more than one entity and are subsequently considered ‘nonspecific’.[16]

Table 1: Showing dermoscopic patterns corresponding to histopathological features in lichen planus and its variants.[17]

	Dermoscopic patterns	**Corresponding histopathological changes**
1.	Pearly white areas (Wickham striae) and peripheral striations	Compact orthokeratosis above zones of wedge-shaped hypergranulosis, acanthosis, and dermal fibrosis
2.	Comedo-like openings	Hypergranulosis and hyperkeratosis of dilated infundibulum
3.	Yellow structures	Spongiosis and vacuolar degeneration of basal cell

Colours play an important role in dermoscopy. Common colours are light brown, dark brown, black, blue, blue-grey, red, yellow, and white. The colour of melanin essentially depends on its localization in the skin.[8]

The colour blue occurs when melanin deposition occurs in deeper dermis because the portions of visible light with shorter wavelengths (blue-violet end of spectrum) are more dispersed than portions with longer wavelengths (red end of visible spectrum).[8]

Table 2: Showing various pigment patterns on dermoscopy corresponding to location of melanin deposits in lichen planus and its variants.[8]

	PIGMENT PATTERNS ON DERMOSCOPY	LOCATION OF MELANIN DEPOSITS
1.	Black	Stratum corneum and the upper epidermis
2.	Light to dark brown	Epidermis
3.	Grey to grey-blue	Papillary dermis
4.	Steel-blue	Reticular dermis

The colour red is seen due to increase in number or dilatation of blood vessels, trauma, or neovascularization. Red dots represent normal papillary vessels, red lines are the deeper ectatic horizontal subpapillary capillaries and linear vessels have been termed as radial capillaries.[18] The colour white is due to regression or scarring.

ETIOPATHOGENESIS

It is evident that Lichen Planus (LP) is an immunological disease, which is thought to represent an abnormal delayed hypersensitivity reaction to an undetermined epidermal neoantigen.[19] T-cell-mediated autoimmune damage to basal keratinocytes that express altered self-antigens on their surface.[20]

An important component for the generation of effector T cells with cytotoxic potential is the presentation of these exogenous antigens in the context of antigen-presenting cells.[20]

Both antigen specific and nonspecific mechanisms are involved in the initiation of immune reaction.[1]

The antigen specific mechanisms involves antigen presentation by basal keratinocytes and antigen specific keratinocyte killing by CD8+ cytotoxic T cells while the nonspecific mechanisms include mast cell degranulation and activation of matrix metalloproteinases (MMP).[1]

T cells play an important role in the pathogenesis of LP. Activated T lymphocytes are recruited to dermoepidermal junction. Both helper T-cells and suppressor T-cells are activated. In the early phase, there is preponderance of CD4+ cells in the dermis, while in the established lesions, predominantly CD8+ cells are seen especially in the epidermis.[19]

Th17 produces interleukin-17 (IL-17) which enhances T-cell-mediated reactions and induces the production of chemokines and other cytokines which leads to production of MMP and promote extracellular matrix (ECM) injury.[21]

Various cytokines that are involved in the pathogenesis of LP include interferon-γ (IFN-γ), tumor necrosis factor-α (TNF-α), additional nuclear factor-κB (NF-κB)-dependent cytokines, such as IL-1 α, IL-6, and IL-8, and other apoptosis-related molecules, such as Fas/2 Apo-1 and Bcl-2.[19]

Epidermal neoantigen, is the yet to be identified antigen, processed by the antigen presenting cell in the epidermis and presented to the T-lymphocytes.

IFN-γ produced by T-helper cells upregulates the expression of intercellular adhesion molecule-1 (ICAM-1) by basal keratinocytes which acts as a ligand for β2-integrin, a leukocyte function associated antigen (LFA) on the surface of lymphocytes, following which lymphocytes and keratinocytes interact, responsible for band-like lymphocytic infiltrate close to the DEJ.

IFN-γ also induces keratinocytes to produce various cytokines such as TNF-α and to upregulate MHC class II, thus increasing the interaction with T-helper cells. Activated CD8+ lymphocytes undergo clonal expansion at the lesional skin. CD8+ lymphocytes and natural killer (NK) cells induces apoptosis of keratinocytes by two different mechanisms resulting in colloid bodies.[1]

The first pathway involves the cross-linking of a surface membrane ligand (Fas ligand) present on antigen-activated cytotoxic T lymphocytes and a death receptor (Fas) on the target keratinocyte. This initiates a cascade, involving proteolytic enzymes ultimately resulting in target cell apoptosis and lysis.[22]

The second which is more important mechanism is via release of cytotoxic granules, such as perforin and granzyme B. Granzymes enter the cytosol of the target cell through hole induced by perforin in the host cell membrane. Granzyme B then induces a direct cytotoxic damage on keratinocyte

either through caspase cascade-dependent or caspase cascade-independent pathways.[1]

The combination of continued keratinocytes destruction and regenerative activity results in development of typical papule.[19]

Target antigens include Hepatitis C virus, vaccines (HBV vaccines), to dental restorative materials (such as amalgam and gold), colour film developers, musk ambrette, nickel, local trauma, plaque causing microorganisms and drugs like beta blockers, diuretics, anti-malarials, penicillamine.[23]

Significant association between specific HLA antigens and LP is seen. An increased frequency of HLA-B27, HLA-B51, HLA-Bw57 (oral LP in English patients), HLA-DR1 (cutaneous and oral LP), HLA-DR9 (oral LP in Japanese and Chinese patients) and HLA-DR6 (HCV-associated).[20]

LICHEN PLANUS

Lichen planus (LP) is a common inflammatory disease affecting the skin, mucous membranes, nails and the scalp.[24] It is characterized clinically by pruritic, polygonal, violaceous flat-topped papules and plaques. The surface appearing shiny with branny scale that forms fine, whitish streaks known as Wickham's striae (WS).[25]

Lichen Planus lesions are typically symmetrical in distribution[26], predominantly over flexural surfaces of upper and lower extremities, trunk and

sacral region. Oral mucosal involvement is most commonly seen. Vulvovaginal, oesophageal, and conjunctival mucosa may also be involved. The face is rarely affected.[25]

Lichen planus affects 0.2% to 1% of the adult population worldwide. LP is commonly seen in adults during their fourth to sixth decade and is rare in children.[5]

The morphological variants are linear, annular, atrophic, hypertrophic, inverse, eruptive, bullous, ulcerative, LP pigmentosus, lichen planopilaris, actinic, lichen planus-lupus erythematosus overlap syndrome and lichen planus pemphigoides.[27]

Histopathology

Epidermal changes characterized by irregular epidermal hyperplasia with a jagged "saw-tooth" appearance, compact hyperkeratosis, wedge shaped focal hypergranulosis, acanthosis with toothing of rete ridges, numerous fibrillar Civatte bodies and basal cell liquefaction.[19]

Dermal changes are characterized by a dense, continuous, band-like inflammatory infiltrate predominantly of lymphocytes with a few macrophages hugging the dermoepidermal junction (DEJ) and pigment incontinence.[19, 28] The inflammatory infiltrate is predominantly perivascular.[19]

LP lesion may resolve with residual hyperpigmentation caused by a persistent increase in the number of melanophages in the papillary dermis.[22]

Direct Immunofluorescence (DIF) reveals a ragged or shaggy fibrin basement membrane zone (BMZ) band. Globular deposition of colloid bodies mainly with IgM and to a lesser extent with IgA, IgG and C3 seen in the upper dermis, around the DEJ and lower epidermis.[1]

Dermoscopic features

WS is the most common feature seen on dermoscopic examination in LP lesions.[29] The other dermoscopic features include grey-blue dots, comedo, milium-like cysts, and vascular structures (red lines).

Different patterns of WS are reticular, Leaf venation, circular, radial streaming, linear, globular, perpendicular, veil-like structureless and a combination of these patterns.[30]

Initial LP lesions show small, round WS centered by a yellow-brown dot, which corresponds to vacuolar alterations of the basal keratinocytes and spongiosis in the spinous zone. In mature LP lesions, WS become polymorphic, showing thin ("comblike" spikes) or broad arboriform projections of the border with disappearance of central yellow-brown areas. Evolved LP lesions show WS whereas in long-standing lesions, pigmented structures, with or without WS seen according to their duration and the intensity of the inflammatory process.[15]

Dotted, globular or linear vessels mainly localised at the periphery of the lesion.[31] In mature LP lesions, prominent peripheral linear, radial capillaries surround the WS contour, which are intermingled with the projections of the border are seen. Characteristic round vessels are less commonly seen. In evolved LP lesions, peripheral vessels are less prominent whereas long-standing LP lesions are devoid of capillaries.[15]

Violet, reddish, pink, brown or yellow background is seen.[32]

Evolved LP lesions show WS and pigmented structures which begin to appear, surrounding the WS contour.[15]

White/ yellow dots and some pigmented structures (dots, globules and/ or reticular or cloud-like areas) are other additional dermoscopic findings of active lesions.[32]

The intense granular deposits indicate slower and persistent course of the disease, whereas diffuse pattern of pigmentation with an absence of globules or dots were associated with earlier resolution.[33]

WS appear more uniform in colour with nonpolarized than with polarized dermoscopy, which may give them an "unfocused" appearance.[15]

Dermoscopy provides useful information for monitoring of pigmented LP, as lesions exhibiting multiple grey-blue-brown granules seem to persist longer than lesions showing brownish structureless areas.[34]

1. Linear Lichen Planus

Linear LP, a rare variant attributes to linear pattern of trauma via the koebner's phenomenon or may follow a dermatomal or blaschkoid pattern. In linear LP, lesions are along blaschko's lines, are usually unilateral, pruritic and may involve any area of the body.[35]

2. Annular Lichen Planus

Annular lichen planus present as red to purple, macules or plaques with raised borders where central atrophy may be seen. The ring shape may develop as a result of convergence of multiple lichenoid papules in a circular shape or expansion of a papule or plaque with central involution and an advancing raised border.[36] They can be either single or multiple.

Annular Atrophic lesions are often asymptomatic or few may present with pruritus. These lesions are commonly present over penis and scrotum.[37] On histopathological examination, epidermal thinning, lichenoid infiltrate and loss of elastic fibers in the center of the active lesions are seen.[38]

Dermoscopy of Annular atrophic lichen planus: In early lesions, peripheral homogeneous vascular patterns and central homogeneous pigmentation is seen. After treatment, vascular patterns disappears but pigment patterns persist.[39]

3. Atrophic Lichen Planus

They appear as well-demarcated white-bluish or brown papules and plaques. Most often seen following the resolution of annular or ulcerative lesions. Common sites include axillae, glans penis, lower extremities and trunk.[37] Histopathology shows loss of rete ridges and dermal fibrosis.[22]

4. Lichen Planus Hypertrophicus (LPH)

They are characterized by thick hyperkeratotic plaques with verrucous surface present symmetrically on anterior aspect of leg or dorsal aspect of the foot, associated with pruritus. The lesions are chronic in nature because of repetitive scratching.[40]

On histopathology

Hyperkeratosis, parakeratosis, hypergranulosis, papillomatosis, acanthosis, hyperplasia, markedly increased thick collagen bundles in the dermis are seen. The rete ridges are more elongated and rounded as opposed to the typical saw-tooth pattern.[41]

Dermoscopy:

In LPH, grey-blue globules and pearly white areas are interspersed and arranged in diffuse structure-less pattern.[17] The lesions may additionally display

yellow structures, comedo-like structures, filled with round corneal masses and diffusely arranged red dots.[42]

Complications:

- Chances of Malignant transformation in long-standing cases.[1]

5. Inverse Lichen Planus

These lesions are usually confined to the intertriginous areas which includes axillae, inguinal creases, gluteal cleft, limb flexures, and sub-mammary region. Clinically presents as erythematous lichenified plaques with poorly defined borders. Scales may be absent due to the occlusive nature of these locations.[43]

6. Eruptive Lichen Planus

Eruptive LP or exanthematous or generalized LP presents as rapidly spreading, disseminated, erythematous to violacous, flat-topped, polygonal papules or macules which resolve with hyperpigmented macules. Lesions may appear over the trunk, all four extremities and the mucosal surface.[37]

7. Bullous Lichen Planus

Bullous LP (BLP) is a rare variant which typically presents as tense bullae on the surface of pre-existing LP lesions. Most common site to be

affected is leg, can also occur over trunk and dorsal aspects of the hands and feet. BLP distinguished from lichen planus pemphigoides using immunofluorescence.[44] Some of the distinctive features such as hyperkeratosis, hypergranulosis, or dense lymphocytic dermal-epidermal infiltrate may not be present as the disease progress quickly.[22] Direct and indirect immunofluorescence (IF) shows negative results.[5]

8. Lichen planus pemphigoides (LPPe)

Lichen planus pemphigoides is a rare autoimmune subepidermal blistering dermatosis. It presents with characteristic features of both classic LP and bullous pemphigoid clinically, histologically and immunopathological features. Clinically presents with tense bullae seen over both involved and uninvolved skin. Lesions commonly occur over the extremities but may occur anywhere, including the oral mucosa.[45] LPPe has also been linked to Castleman's disease and various neoplasms, including retroperitoneal round-cell liposarcoma, chronic lymphocytic leukemia, and other soft-tissue tumors.[45, 46]

On DIF, Peribullous skin shows linear deposition of IgG and C3 along the dermal–epidermal junction.[20] Circulating autoantibodies against BMZ components are often found using indirect immunofluorescence (IIF).[1] Immunoelectron microscopic studies reveal deposition of IgG and C3 in the base of the bulla and not in the roof as found in bullous pemphigoid.[5]

9. Ulcerative or erosive variant of Lichen Planus

This variant of LP may be found on mucosal surfaces, but also occurs on the plantar surface of the feet. Ulcerative LP presents clinically as ill-defined, chronic, painful ulcers on the plantar surface resulting in difficulty in walking. Perilesional skin can be erythematous and scaly with loss of toenails.[37]

10. Lichen planus-lupus erythematosus

Lichen planus-lupus erythematosus overlap syndrome is a rare variant, which presents with features of both LP and lupus erythematosus in the same patient or in the same lesion. Common sites include face, trunk, distal arms and legs. DIF may be essential in diagnosing this overlap syndrome.[47]

11. Palmoplantar Lichen Planus

Clinically presents as erythematous, scaly plaques, associated with itching, with or without palmoplantar keratoderma, characteristically involving the internal plantar arch and palms with sparing of the fingertips. Histopathological examination is essential to differentiate palmoplantar LP from other conditions which show findings similar to classic LP.[48]

12. Erythrodermic Lichen Planus

Erythrodermic Lichen Planus is very rarely seen and documented in the literature. Morphologically, erythematous or violaceous papules and plaques

with extensive involvement of the body surface, with or without scaling are seen. Among the erythroderma, typical LP lesions, blisters or erosions may appear in a localized fashion. Pruritus is severe and the general health is altered.[43]

Table 3: Differential Diagnoses of Different Variants of LP [1, 23]

Classic	Psoriasis Drug eruption Lichen simplex chronicus
Annular	Granuloma annulare Tinea
Linear	Nevus unius lateris Lichen striatus Linear epidermal nevus
Hypertrophic	Lichen simplex chronicus Prurigo nodularis Lichenoid cutaneous amyloidosis Kaposi sarcoma Warts
Atrophic	Lichen sclerosis et atrophicans Morphea

Follicular	Lichen nitidus Lichen spinulosus
Guttate LP	Guttate psoriasis Pityriasis lichenoides chronica Papulosquamous secondary syphilis
LP pemphigoides	Bullous LP Bullous Pemphigoid
Palmoplantar LP	Callosities Warts Secondary syphilis

TREATMENT OF LICHEN PLANUS

1. Topical therapy :
    - Potent to highly potent topical steroids.[49]
    - Vitamin D analogues: calcipotriol.[49]
    - Calcineurin inhibitors: topical tacrolimus, pimecrolimus.[50]
    - Intralesional corticosteroids injection.[49]
    - Retinoids: Isotretinoin, Retinoic acid.[51]
2. Systemic therapy-
    - Systemic corticosteroids.[49]

- Systemic retinoids: Acitretin, Isotretinoin, All-trans-retinoic acid, 13-cis-retinoic acid, Etretinate (Combination of PUVA and retinoids).[51]
- Cytotoxic drugs: Methotrexate,[52] Azathioprine.[53]
- Anti- malarials: Hydroxychloroquine.[54]
- Immunomodulatory drugs: mycophenolate mofetil, [55] Cyclosporine,[56] thalidomide [57]
- Anti- epileptics: Phenytoin.[58]
- Anti- fungals: Griseofulvin,[59] Itraconazole.[49]
- Dapsone (oral and topical).[60]
- Sulfasalazine.[61]
- Metronidazole.[62]
- low molecular weight heparin: Enoxaparin.[63]
- dexamethasone pulse therapy.[49]
- Biologicals- Alefacept, Anakinra, Efalizumab, Adalimumab.[51, 64]

3. Phototherapy
    - Ultraviolet (UV) phototherapy- UVB and PUVA therapy.[65]
    - ALA-mediated topical photodynamic therapy.[66]

ORAL LICHEN PLANUS

Oral lichen planus may appear alone or in combination with cutaneous LP. Oral LP is classified into six types: reticular, papular, plaque-like, erosive, atrophic, and bullous. The lesions are mostly seen on the buccal mucosa followed by lateral margins of the tongue, gingiva, lips, and hard palate.[67]

Differential diagnosis

- Leukoplakia
- oral pemphigus.[1]

Treatment:

- Topical and oral glucocorticoids[68]
- Topical calcineurin inhibitors[69]
- Systemic corticosteroids[68]
- Anti- malarials: Hydroxychloroquine[70]
- topical human fibroblast interferon (HuIFN-β) and interferon-α (IFN-α).[55]
- 308 nm excimer laser[71]
- New biologic therapeutic agents such as alefacept, basiliximab, efalizumab and etanercept.[55]
- Newer, topical rapamycin (now known as sirolimus) and extracorporeal photochemotherapy for erosive oral lichen planus.

peroxisome proliferator– activated receptor agonist for lichen planopilaris; and anti-CD20 monoclonal antibody.[72]

Complications

- Chances of Malignant transformation in long-standing cases.[1]

NAIL LICHEN PLANUS

Nail involvement affects up to 10% of patients with LP lesions involving other sites. Nail LP may sometimes be the only manifestation of the condition. Affected nails present with longitudinal ridges, pitting, pterygium, onychorrhexis, trachyonychia, pup-tent nail and onycholysis.[73]

Differential diagnosis

- Onychomycosis,
- Nail psoriasis
- Alopecia areata[1]

Treatment

- Topical retinoids: Tazoretene
- Calcineurin inhibitors :Topical tacrolimus
- Topical corticosteroids
- Topical 5 Flurouracil

- Intralesional steroid injection
- Systemic corticosteroids- intramuscular injections and oral glucocorticoids
- Systemic retinoids : Alitretinoin, Etretinate
- Low- dose Methotrexate
- Cyclosporine[74, 75]

GENITAL LICHEN PLANUS

It is an uncommon variant that involves vulva and vagina. Usually presents as a chronic course, with unexplained exacerbations, improvements and remissions with complaints of genital irritation, itching, burning, soreness, purulent discharge and dyspareunia. On physical examination, inflamed, erythematous, and/or eroded epithelium may be seen which may transform into reticulated white-grey lacy pattern.[37]

Male genitalia are involved in 25% of cases and the glans penis is most commonly affected, with annular lesions as frequent presentation.[23]

Differential diagnosis

Female

- Leukoplakia,
- Lichen sclerosus et atrophicus

Male

- Psoriasis
- Lichen sclerosus et atrophicus[1]

Treatment:

- Potent topical corticosteroids
- Calcineurin inhibitors: topical tacrolimus
- Systemic corticosteroids
- Other drugs: Azathioprine, Mycophenolate mofetil, Hydroxychloroquine, Cyclosporin, Methotrexate, Retinoids, Thalidomide
- Photochemotherapy[76, 77]

ACTINIC LICHEN PLANUS (ACTINIC LP)

Actinic LP also known as lichen planus subtropicus, is a rare variant that presents as asymptomatic lesions, affecting sun-exposed areas which include forehead, cheeks and lips, the upper chest, extensor surface of distal forearms and dorsum of hands. Eruptions occur more often during spring and summer and remission during winter months.

Three clinical variants are seen i. e. annular, pigmented and dyschromic. Annular actinic LP is the most common form, characterized by annular

erythematous brownish plaques, with or without atrophy. Pigmented forms present as hypermelanotic patches with melasma-like appearance. Dyschromic type, as rarest type presents with whitish pinhead coalescing papules.[37]

Dermoscopic examination:

Wickham striae (WS) pattern is not observed even in early active lesions.[39]

Differential diagnosis

- Polymorphic light eruptions[1]

Treatment

Sun protection, topical corticosteroids, hydroxychloroquine, cyclosporine and acitretin.[58, 78]

LICHENOID DRUG ERUPTION (LDE)

Lichenoid drug eruption (LDE) resembles LP on clinical and histological basis, commonly seen in adults, approximately 10 years older than those with idiopathic LP. Both the genders are equally affected. There is usually a latent period of several months from drug introduction to the appearance of the cutaneous eruption.[20]

LDE tends to occur in chronically sunexposed areas such as face rather than on forearm and the lesions are more generalized. The individual lesion may look like LP or may be more eczematous, psoriasiform or pityriasiform in appearance. Classical WS is absent and generally oral mucosa is not involved. There is greater risk of developing post inflammatory hyperpigmentation. The drugs causing LDE are angiotensin converting enzyme inhibitors, thiazide diuretics, gold, antimalarials, penicillamine, nonsteroidal anti‑inflammatory agents, dental amalgams, sulfasalazine, β‑blockers, and proton‑pump inhibitors.[79]

Histopathology

LDE characteristically exhibit parakeratosis, a dermal eosinophilic and plasma cell infiltrate and a perivascular lymphocytic infiltrate affecting the reticular dermis. Epidermal changes are less commonly seen in lichenoid drug eruptions when compared to classic LP. However, increased necrotic

keratinocytes with eosinophilic infiltration can be helpful in distinguishing lichenoid drug reaction from cutaneous LP.[80]

DIF reveals globular deposition of colloid bodies with IgM and occasionally with IgA, IgG and C3 seen in the upper dermis, around the DEJ and lower epidermis with fibrin at DEJ.[1]

Treatment: Withdrawl of the offending drug, topical and systemic steroids.[1]

LICHEN PLANUS PIGMENTOSUS (LPP)

Lichen Planus Pigmentosus (LPP) presents with asymptomatic, slate grey to brownish black macules on sun-exposed sites including the face, neck, upper extremities and over flexures.

LPP typically presents in individuals with skin types III and IV, around fourth to fifth decade.[20]

Pigment patterns within lesions are commonly arranged in diffuse pattern, while blotchy, reticular, perifollicular and unilateral linear patterns are rare. Scalp, nail, or mucosal involvement is rare. LPP usually has a longer clinical course than other variants of LP.[81]

The common differential diagnosis is Erythema dyschromicum perstans.[1]

Dermoscopic examination:

It is characterised by the presence of pigment pattern like slate grey-to-blue dots and globules, perifollicular and peri-eccrine pigment deposition. Hem-like pigment pattern with brown colour background on dermoscopy is seen.[82]

Even in the active and early phase, Wickham striae (WS) patterns and vascular patterns are not observed in LPP.[39]

In LPP lesions, perifollicular/annular, linear, and cobblestone pigment patterns are seen both initially and after treatment.[83]

In the early phase 'diffuse peppering' pattern is seen which changes to "reticular" pattern followed by appearance of "perifollicular/annular" pattern that manifests as half circular fine pigmentation instead of annular dark pigmentation.[39]

In some LPP lesions, the pigment pattern may be absent in skin furrows, as they are not exposed to friction, which could be the reason for the absence of pigmentation.[39]

In a dermoscopic study done on LPP, three novel dermoscopic signs were seen which includes brownish ovoid nests, pigmented targetoid globules and bluish black fine dots.[84]

Histopathological examination

LPP follows the typical histopathological changes as seen in LP, but with thinning of the epidermis.[85]

On IF, IgM is seen and less commonly IgG, C3 and fibrinogen deposits in the colloid bodies. Linear IgM and C3 deposits along the basement membrane zone are seen.[86]

Treatment

1. Topical agents
    - Calcineurin inhibitors: Tacrolimus
    - Hydroquinone
    - Retinoic acid
    - Corticosteroids
    - Azelaic acid
    - Kojic acid
    - Glycolic acid
    - Arbutin
    - 10% aqueous solution of dimethyl sulfoxide.[86, 87]
2. Systemic treatment
    - Systemic corticosteroids
    - Vitamin A
    - Dapsone
    - Griseofulvin
    - Etretinate

- Chloroquine[86, 88]

3. LASER: Q-switched Nd-YAG (1064 nm) [89]

LICHEN PLANOPILARIS (LPp)

It is also known as follicular form of lichen planus, a rare inflammatory lymphocyte-mediated disorder that selectively involves hair follicles causing follicular destruction and gradually leads to cicatricial alopecia.

Clinically, presents with solitary or multiple areas of baldness over scalp commonly involving parietal area and vertex. The alopecia patches expand centrifugally and rarely involve the entire scalp. The most common signs and symptoms are increased hair shedding, pruritus, scaling and scalp tenderness. These symptoms may worsen with exposure to ultraviolet light, sweating and stress. Three groups of LPp seen are classic, frontal fibrosing alopecia and Graham- Little-Piccardi-Lassueur syndrome.[90]

Differential diagnosis

On the skin:

- Lichen nitidus
- Lichen spinulosus

On the scalp:

- Discoid Lupus Erythematosus

- Cicatricial pemphigoid
- Postinflammatory folliculitis
- Keratosis follicularis spinulosa decalvans
- Alopecia areata
- Lupus erythematosus[1, 23]

Dermoscopic features

Trichoscopy reveals multiple areas of irregular cicatricial alopecia with perifollicular whitish-grey scaling associated with erythema, arboriform vessels, follicular plugging and absence of follicular openings.[83]

In LPp, the inflammatory phenomenon usually affects the hair follicles in a selective manner with respect to the interfollicular epidermis.[90]
Dermoscopic findings vary according to the stage of evolution and the degree of disease activity.[83]

In early stages, perifollicular inflammation leads to the appearance of whitish-grey scales (peripilar casts) associated with perifollicular erythema and arboriform vessels.[83]

In the fibrotic stage, whitish or milky-red areas, covered by “classic irregular whitish dots” are seen (fibrous tracts seen as a result of a variable loss of follicular units). Additionally, there are blue-violet areas and blue-grey dots

reflecting perifollicular pigment incontinence. Here, grey-blue globules appear as "target" pattern.[83]

Histopathology

Band-like lymphocytic infiltrate is initially seen in the peribulge area, infundibulum and isthmus with the sparing of lower segment of the hair follicle. The follicular segment may show orthokeratosis, hypergranulosis, and follicular plugging. The interfollicular epidermis is rarely affected.[91]

IF shows IgM, IgG and IgA are found in varying combinations along the follicle–dermal interface.[20]

Treatment

1. Topical therapy: High-potency topical corticosteroids.[92]
2. Intralesional injections with triamcinolone acetonide.[92]
3. Systemic therapy:
    - Systemic corticosteroids[92]
    - Systemic retinoids (acitretin or isotretinoin)[92]
    - Anti- malarials: Hydroxychloroquine[93]
    - Cytotoxic drugs: Methotrexate,[94] Azathioprine
    - Immunomodulatory drugs: Mycophenolate Mofetil,[95] Cyclosporine,[96] thalidomide [97]

- Pioglitazone[95]
- Griseofulvin[95]
- Laser therapy: 308-nm excimer[95]

LICHEN NITIDUS

Lichen nitidus presents with multiple, small, discrete, flat topped, shiny skin colored to slightly pink papules that may occur anywhere on the skin.

It is more prevalent among children or young adults, and a female predominance has been described in the generalized variant.[20]

The most common sites are glans, shaft of the penis, abdomen, and extremities. Involvement of mucous membrane, nails, palms, and soles are rarely seen. The other morphological variants include generalized, vesicular, linear, perforating, spinous, follicular, hemorrhagic and actinic.[98]

Differential diagnosis

- Lichen planus
- Psoriasis
- Keratosis pilaris
- Lichen spinulosus
- Lichen scrofulosorum
- Verruca plana[1, 23]

Histopathology

Epidermis is thinned out with thickened parakeratotic horny layer and hydropic degeneration of basal keratinocytes with occasional civatte bodies. A dense, well-circumcised subepidermal infiltrate enclosed by a ‘‘claw-like’’ rete ridges filling the space of one to five dermal papillae.

In the early stages, lymphocytic infiltration is seen which is later replaced by granulomatous infiltrate with occasional giant cells.[99] DIF studies in lichen nitidus have given negative results.[5]

Treatment

- Oral and topical steroids
- Topical calcineurin inhibitors
- Astemizole
- Systemic retinoids: Isotretinoin, Acitretin
- Low-dose cyclosporine
- Phototherapy
- Itraconazole
- Isoniazid[100, 101]

LICHEN STRIATUS (LS)

Lichen Striatus is an acquired, self-limiting condition, characterised by asymptomatic, small, pinpoint to 2mm flat-topped pink or skin-coloured monomorphic papules, coalescing into a linear band-like configuration along blaschko's lines.

Lichen striatus usually occur as isolated lesions on the limbs in children aged 5–15 years and generally resolve over 6–12 months. Females are affected approximately two to three times as frequently as males.[5]

The cause of LS is unknown and it is hypothesize that, its pathogenesis involves a combination of genetic and environmental factors. Distribution of LS along blaschko's lines suggests that post-zygotic somatic mosaicism or mutation of epidermal progenitor cells which occurs during embryogenesis. The lesions are usually asymptomatic and rarely may be pruritic, present unilaterally on the extremities, less frequently present over head, neck, and trunk. Post-inflammatory hypopigmentation after resolution can be seen.[102]

Dermscopic features

A case of Lichen striatus was reported with dermoscopic finding as deep-whitish structures which resembled white scar-like areas with "cerebriform or nutmeg apearence."[103]

In a study, the significant dermoscopic feature of LS was grey granular pigmentation which histologically corresponded to pigment- laden dermal melanophages of LS.[104]

Histopathology

Epidermal features include dyskeratosis, spongiosis, and exocytosis. A lichenoid inflammatory infiltrate near the DEJ, infiltration of lymphocytes and histiocytes in perivascular area in upper dermis, which may extend to deep dermis are occasionally seen. A characteristic feature is the presence of dense peri-eccrine and periadnexal lymphocytic infiltrate.[105] DIF has shown negative results in LS.[1]

Treatment

- Topical corticosteroids
- Topical tacrolimus and pimecrolimus
- Cyclosporine[106]

ERYTHEMA DYSCHROMICUM PERSTANS (EDP)

Erythema dyschromicum perstans is characterized by asymptomatic, slowly progressing, ash-grey, macular hyperpigmentation of the skin. The

hallmark is the slightly raised, erythematous border in the initial inflammatory stage.

It is commonly seen in individuals with skin types III and IV and in women when compared to men. EDP frequently appears during the first to third decade of life.[20]

In pigmentary stage, macules in various shades of grey are observed mainly on the trunk, limbs, and face.[107]

In the active lesions, vacuolar degeneration of basal cells, pigmentary incontinence in upper dermis and perivascular lymphohistiocytic infiltrate are seen. In the residual macules, pigment incontinence are seen predominantly whereas minimal to intense cellular infiltrate and basal cell vacuolar degeneration are seen.[108]

DIF microscopy studies of the active border have demonstrated IgM, IgG, fibrinogen and C3 staining of colloid bodies.[20] Treatment includes corticosteroids, vitamins, antihistamines, chloroquine, clofazimine, dapsone and isotretinoin.[108]

KERATOSIS LICHENOID CHRONICA (KLC) OR NEKAM'S DISEASE.

Clinically, lichenoid keratotic papules develop in parallel linear or reticulate patterns symmetrically on the extremities and lumbosacral region. Facial involvement follows and resembles seborrheic dermatitis or rosacea in

75% of adults. In the pediatric population, lesions typically begin with a purpuric macular eruption on the face that becomes hyperpigmented.[109] It occurs commonly in women around fifth or sixth decade of life.[1]

On histopathology, epidermal changes including alternating areas of atrophy and acanthosis with focal parakeratosis, follicular plugging, lichenoid lymphocytic reaction pattern with focal basal vacuolar alteration. Perivascular and periappendageal lymphocytic and few plasma cells infiltrates are present.[110] Topical corticosteroids, calcpotriol, salicylic acid, systemic steroids, methotrexate, anti-malarials has not provided satisfactory results whereas acitretin and phototherapy has shown better improvement.[111]

CONDITIONS ASSOCIATED WITH LICHEN PLANUS

- Alopecia areata
- Autoimmune thyroiditis
- Myasthenia gravis
- Lichen sclerosus
- Vitiligo
- Discoid Lupus Erythematosus
- Hepatitis C Virus infection
- Thymoma
- Laugier–Hunziker syndrome

- Primary biliary cirrhosis
- Primary sclerosing cholangitis
- Ulcerative colitis
- Diabetes mellitus
- Autoimmune polyendocrinopathy.[1, 112]

SYNDROMES ASSOCIATED WITH LICHEN PLANUS

Graham-Little-Piccardi-Lasseur syndrome

It is a type of lichen planopilaris, which is characterized by a triad of patchy cicatricial alopecia of scalp, noncicatricial alopecia of axilla and groin and follicular spinous papule on body, scalp or both.[113]

Grinspan's Syndrome

Association of Oral Lichen Planus with Diabetes Mellitus and Hypertension is called Grinspan's syndrome.[114]

Vulvovaginal-gingival syndrome

The association of lichen planus of vulva and vagina with desquamative gingivitis is described as vulvovaginal-gingival syndrome.[115]

METHODOLOGY

METHODOLOGY

The present study was carried out in the Department of Dermatology at R.L Jalappa Hospital attached to Sri Devaraj Urs Medical College, Tamaka, Kolar from January 2017 to July 2018. A total of one hundred patients who presented with lichenoid dermatoses and those who satisfied the inclusion criteria were included in this study.

The present study was an observational study.

Inclusion criteria:

All new patients presented with lichenoid dermatoses were included in the study.

Exclusion criteria:

1. Patients who were receiving treatment one month prior to the study.
2. Patients with secondary infection superseding Lichenoid dermatoses.

Methods of data collection:

All cases with lichenoid dermatoses were screened and those satisfying the inclusion criteria were included in the study. After an informed consent, a detailed history regarding symptoms, duration and drug history was taken.

Cutaneous examination and scalp examination were done. Mucosal and nail changes were noted.

The lesions were examined directly through the dermoscope Dermlite DL3N with 10x magnification using both polarized and non-polarized mode.

Then, the dermoscopic photographs were taken with the help of an adaptor attached to an iphone 8 camera with a 12x zoom.

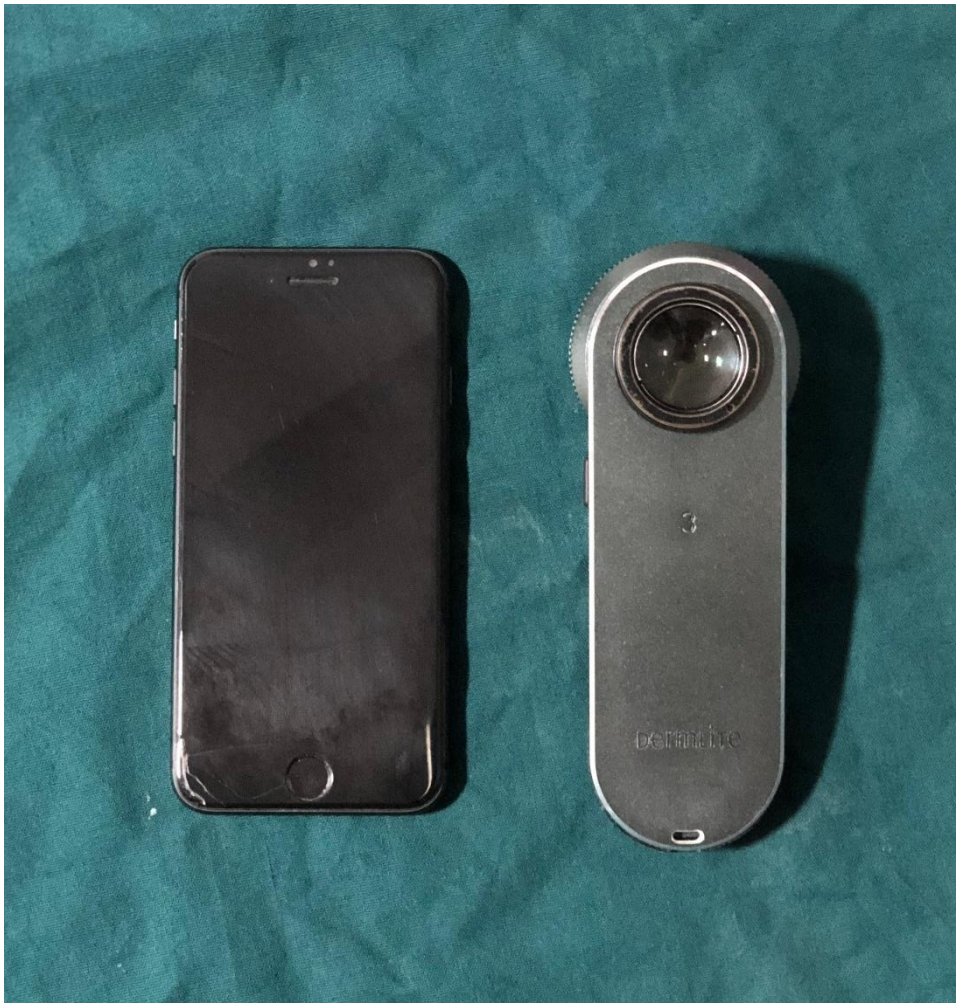

Figure 8: showing iphone 8 with Dermlite 3N dermoscope

The dermoscopic features include wickham's striae (WS), morphology and colour of WS, background colour, pigment patterns (grey-blue and brown dots), vascular structures, comedo like openings, milium-like cysts, pearly white striations, structureless areas, loss of follicles and perifollicular scaling and cast.

STATISTICAL METHODS USED FOR DATA ANALYSIS

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Chi-square was used as test of significance. Continuous data was represented as mean and standard deviation.

Graphical representation of data: MS Excel and MS Word was used to obtain various types of graph such as bar diagram and pie diagram.

p value (probability that the result is true) of <0.05 will be considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel, SPSS version 22(IBM SPSS Statistics, Somers NY, USA) was used to analyse the data.

Sample size calculation :

Sample size was estimated by using the proportion of classical lichen planus in a study done on lichenoid dermatoses as 30%.[18]

$$\text{Sample size} = \frac{Z_{1-\alpha/2}{}^2\, p(1-p)}{d^2}$$

Here

$Z_{1-\alpha/2}$ = Is standard normal variate (at 5% type 1 error ($P<0.05$) it is 1.96 and at 1% type 1 error ($P<0.01$) it is 2.58). As in majority of studies P values are considered significant below 0.05 hence 1.96 is used in formula.

p = Expected proportion in population based on previous studies or pilot studies.

d = Absolute error or precision – Has to be decided by researcher.

P = 30 or 0.30

q = 70 or 0.70

d = 10% or 0.10

Using the above values at 95% Confidence level the estimated sample size was 81 subjects and a total of 100 subjects with Lichenoid dermatoses were included in the present study.

RESULTS

RESULTS

The present study was carried out in the Department of Dermatology at R.L Jalappa Hospital attached to Sri Devaraj Urs Medical College, Tamaka, Kolar from January 2017 to July 2018. A total of one hundred patients who presented with lichenoid dermatoses and those who satisfied the inclusion criteria were included in this study. A detailed history and examination of patients was done in all the cases.

Table 4: Showing total number of cases of lichenoid dermatoses

CASES	No of cases	%age
Lichen Planus	45	45
Lichen Planus hypertrophicus	5	5
Actinic Lichen Planus	2	2
Lichenoid drug eruption	1	1
Lichen planus pigmentosus	5	5
Lichen planopilaris	7	7
Lichen nitidus	27	27
Lichen striatus	8	8
Total	100	100

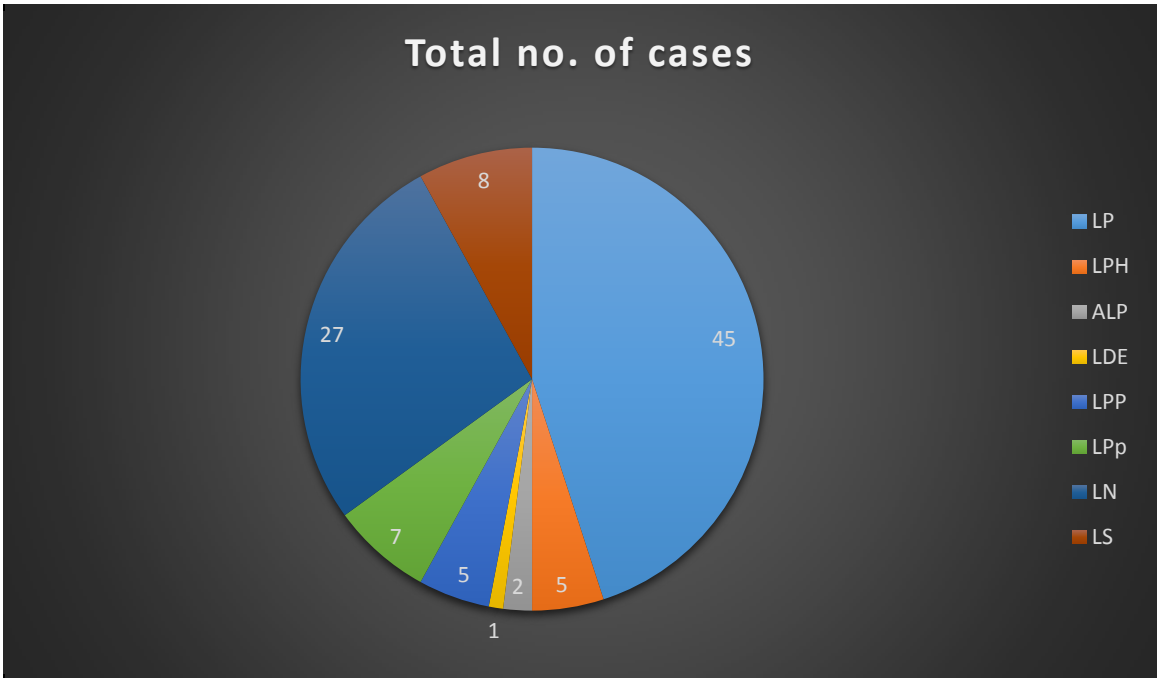

Figure 9: Pie diagram showing total number of cases of lichenoid dermatoses

AGE DISTRIBUTION

In this study, the maximum number of cases 23% were seen in age group of 0-10 years, and the minimum of cases 1% were noted in the age group of more than 80 years. The mean age was 29.34+/- 19.48 years, range varies from 0.9-84 years. (Table 5, Figure 10)

Table 5: Representing age distribution of the population

AGE IN YEARS	N	%
0-10	23	23
11- 20	15	15
21- 30	17	17
31- 40	16	16
41- 50	15	15
51- 60	8	8
61-70	2	2
71- 80	3	3
>80	1	1
TOTAL	100	100%

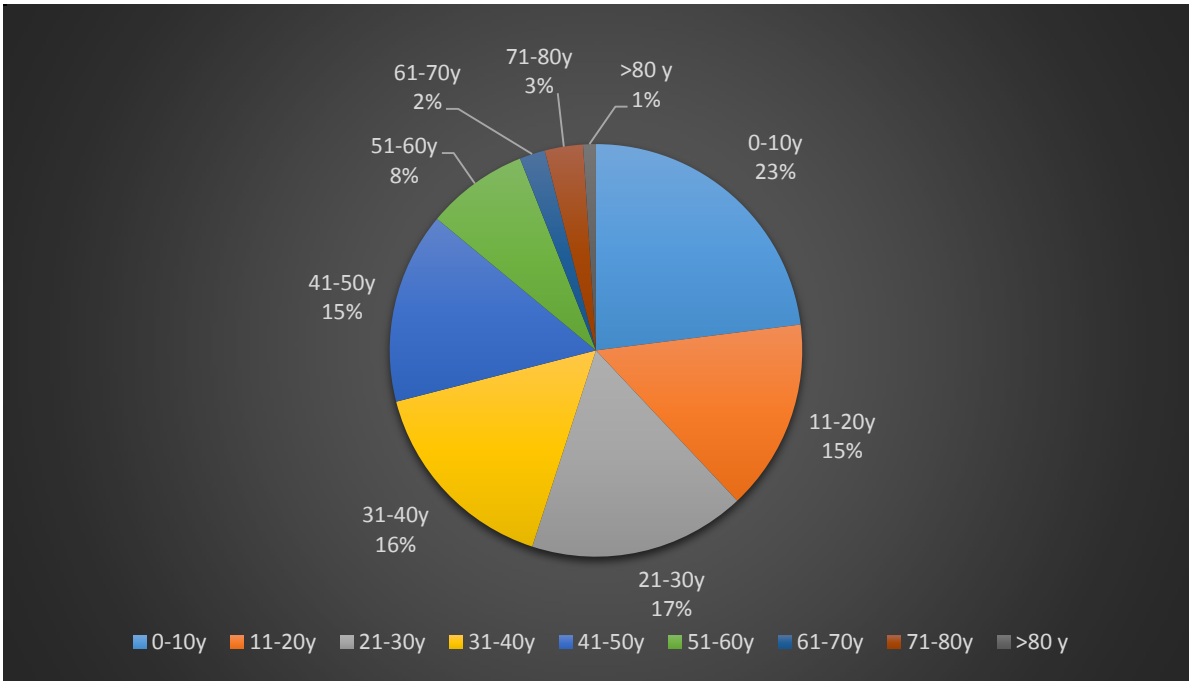

Figure 10: Pie diagram showing age distribution of the population

GENDER DISTRIBUTION

Out of the 100 patients, 51 patients (51%) were females and 49 patients (49%) were males. (Table 6, Figure 11)

Table 6: Gender distribution of lichenoid dermatoses

	No of cases	%age
Male	49	49
Female	51	51
Total	100	100

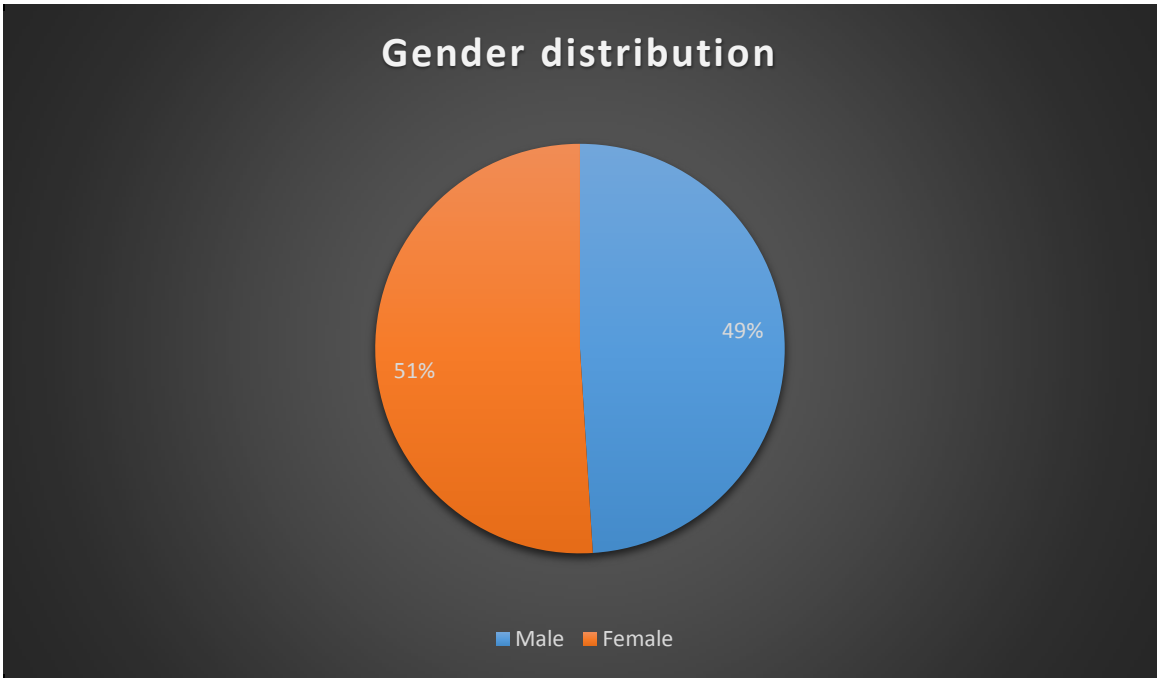

Figure 11: Pie diagram showing gender distribution of lichenoid dermatoses.

EVALUATION OF LICHENOID DERMATOSES IN SUBJECTS SCREENED

Symptoms/ Complaints: 100% of patients with lichen planus and lichen planus hypertrophicus complained of itching, 100% of patients with lichen planus pigmentosus, lichen nitidus and lichen striatus were asymptomatic, 85.7% patients with lichen planopilaris had patchy hair loss over scalp as in table 7 and figure 12.

Duration of lesions: Patients with Lichenoid drug eruption(100%), Lichen nitidus(74%) and Lichen planus(71%) gave 0-3 months history of duration of lesions, whereas Lichen planopilaris(42.8%), Lichen planus pigmentosus(40%) and Lichen planus hypertrophicus (40%) patients gave a history of more than 12 months duration of lesions as mentioned in table 4. The mean duration of lesions was 11.8 +/- 36.66. Minimum duration of lesions was 3 days and maximum duration of lesions was 20 years. (Table 7)

Table 7: Symptoms/ complaints and duration in lichenoid dermatoses

	LP (n= 45)		LPH (n=5)		ALP (n=2)		LDE (n=1)		LPP (n=5)		LPp (n=7)		LN (n=27)		LS (n=8)	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
SYMPTOMS/ COMPLAINTS																
- PRURITUS	45	100	5	100	2	100	1	100	-	-	-	-	-	-	-	-
- BURNING SENSATION	-	-	-	-	2	100	-	-	-	-	-	-	-	-	-	-
- PAIN	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
- ASYMP-TOMATIC	-	-	-	-	-	-	-	-	5	100	1	14.2	27	100	8	100
- LOSS OF HAIR	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
DURATION (in months)																
0-3	32	71.1	3	60	1	50	1	100	2	40	1	14.2	20	74.1	3	37.5
4-12	10	22.2	-	-	1	50	-	-	1	20	3	42.8	4	14.8	2	25
More than 12	3	6.6	2	40	-	-	-	-	2	40	3	42.8	3	11.1	3	37.5

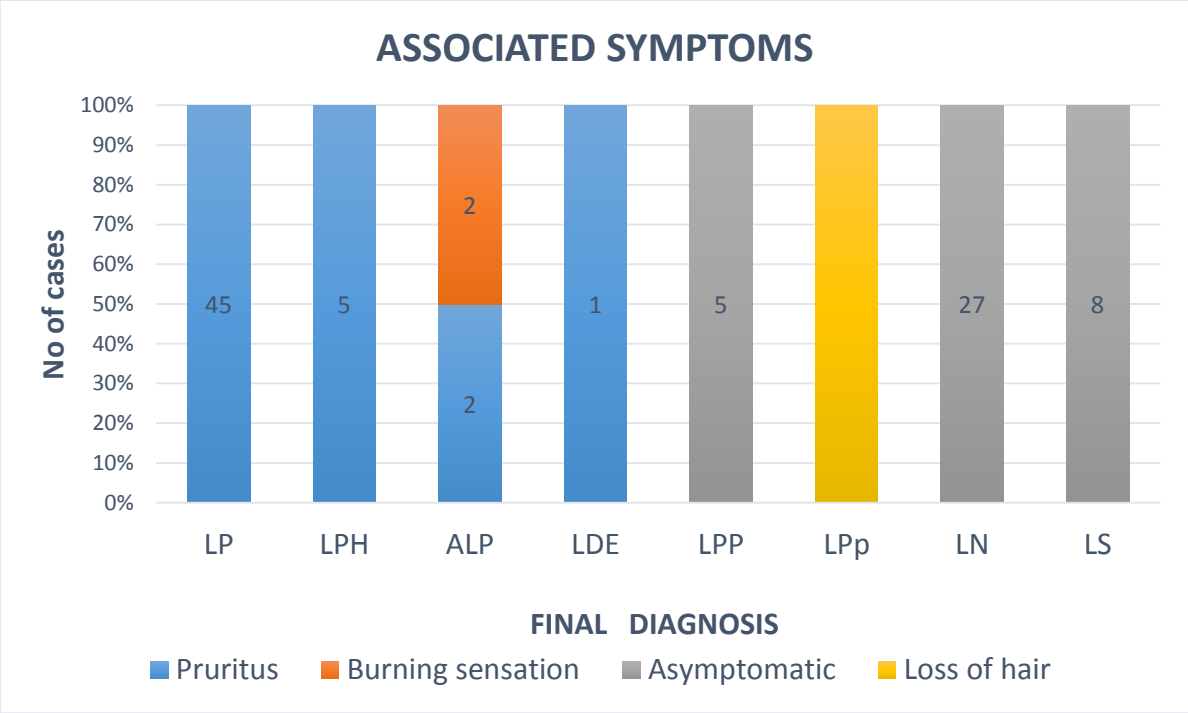

Figure 12: Bar diagram showing associated symptoms according to final diagnosis

ON EXAMINATION

Type of lesions: Lichen planus (64.4%), Lichen nitidus (100%), Lichen striatus (100%) presented as papules, Lichen planus hypertrophicus (60%) as plaques, Lichen planus pigmentosus(100%) as macules and Lichen planopilaris (85.7%) as loss of hair (Table 8a, Figure 13).

Site of involvement: Scalp was involved in 85.7% cases of Lichen planopilaris, face was involved in 100% cases of Lichen planus pigmentosus, upper limb was involved in Lichen nitidus (88.8%), Lichen striatus (62.5%), Lichen planus (62.2%) and Lichen planus pigmentosus (60%). Lower limb was involved in Lichen planus hypertrophicus (100%) and Lichen planus (40%) (Table 8a, Figure 14).

Distribution: Lesions were bilaterally symmetrical in Lichen planus (86.6%), Lichen planus hypertrophicus (100%) and Lichen nitidus(96.2%) whereas unilateral Lichen planus lesions were seen in 6.6% on each side, Lichen striatus on right side in 62.5% cases and on left side in 37.5% cases. Lichen planopilaris had assymetrical distribution in 71.4% cases (Table 8b).

Oral mucosa: In Lichen planus (64.4%) and Lichen planus hypertrophicus (80%) oral mucosa was involved.

Genital mucosa: In 11.1% cases of lichen planus, involvement of genital mucosa was seen.

Nail involvement: Lichen planus showed 26.6% cases of nail involvement.

Table 8a: Distribution of findings on clinical examination

	LP (n= 45)		LPH (n=5)		ALP (n=2)		LDE (n=1)		LPP (n=5)		LPp (n=7)		LN (n=27)		LS (n=8)	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
TYPE OF LESION																
- MACULE	-	-	-	-	-	-	1	100	5	100	2	28.5	-	-	-	-
- PAPULE	29	64.4	-	-	-	-	-	-	-	-	1	14.2	27	100	8	100
- PLAQUE	3	6.6	3	60	2	100	-	-	-	-	-	-	-	-	-	-
- PAPULE+ PLAQUE	12	26.6	2	40	-	-	-	-	-	-	-	-	-	-	-	-
- ANNULAR PLAQUE	1	2.2	-	-	-	-	-	-	-	-	-	-	-	-	-	-
- ALOPECIA	-	-	-	-	-	-	-	-	-	-	6	85.7	-	-	-	-
SITE																
- SCALP	-	-	-	-	-	-	-	-	-	-	6	85.7	-	-	-	-
- FACE	1	2.2	1	20	1	50	-	-	5	100	-	-	3	11.1	1	12.5
- TRUNK	19	42.2	2	40	1	50	1	100	1	20	2	28.5	5	18.5	1	12.5
- UPPER LIMB	28	62.2	1	20	-	-	1	100	3	60	-	-	24	88.8	5	62.5
- LOWER LIMB	40	88.8	5	100	-	-	1	100	1	20	2	28.5	10	37	2	25

Table 8b: Distribution of findings on clinical examination

	LP (n= 45)		LPH (n=5)		ALP (n=2)		LDE (n=1)		LPP (n=5)		LPp (n=7)		LN (n=27)		LS (n=8)	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
DISTRIBUT-ION																
BILATERAL	39	86.6	5	100	-	-	1	100	4	80	2	28.5	26	96.2	-	-
UNILATERAL																
RIGHT	3	6.6	-	-	2	100	-	-	1	20	5	71.4	1	3.7	5	62.5
LEFT	3	6.6	-	-			-	-	-	-			-	-	3	37.5
ORAL INVOLVEMENT																
PRESENT	29	64.4	4	80	-	-	-	-	-	-	-	-	-	-	-	-
ABSENT	16	35.5	1	20	2	100	1	100	5	100	7	100	27	100	8	100
GENITAL INVOLVEMENT																
PRESENT	5	11.1	-	-	-	-	-	-	-	-	-	-	-	-	-	-
ABSENT	40	88.8	5	100	2	100	1	100	5	100	7	100	27	100	8	100
NAIL INVOLVEMENT																
PRESENT	12	26.6	-	-	-	-	-	-	-	-	-	-	-	-	-	-
ABSENT	33	73.3	5	100	2	100	1	100	5	100	7	100	27	100	8	100

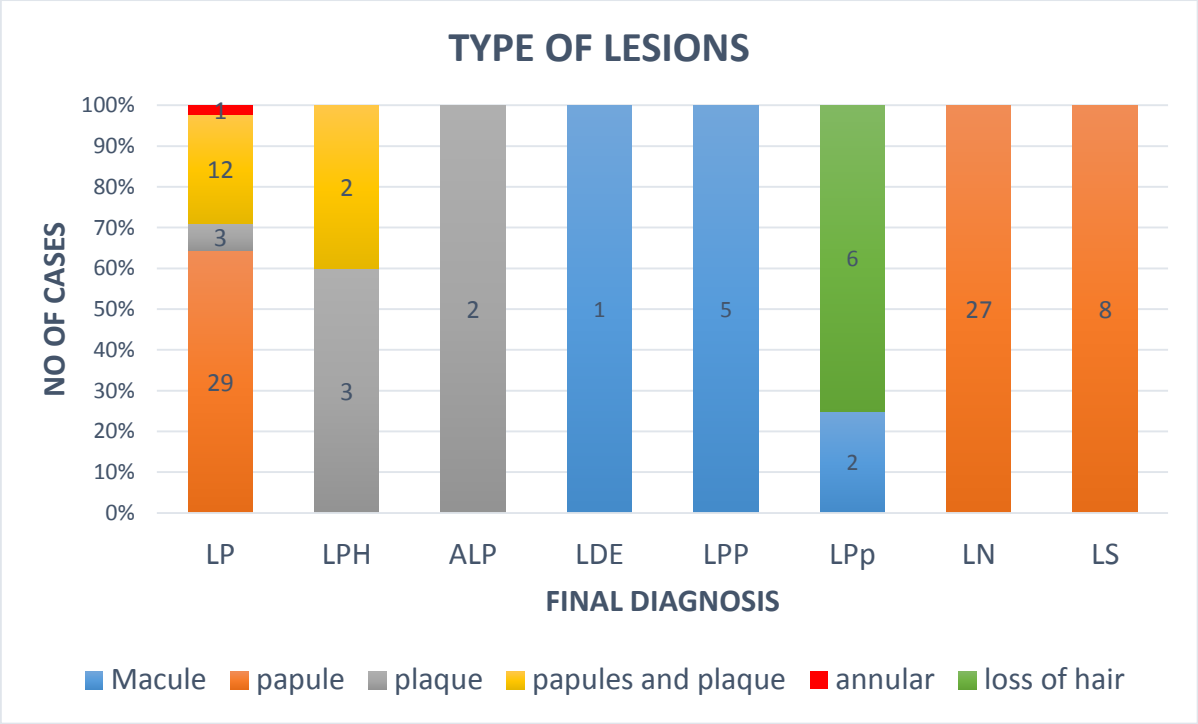

Figure 13: Bar diagram showing type of lesions according to final diagnosis

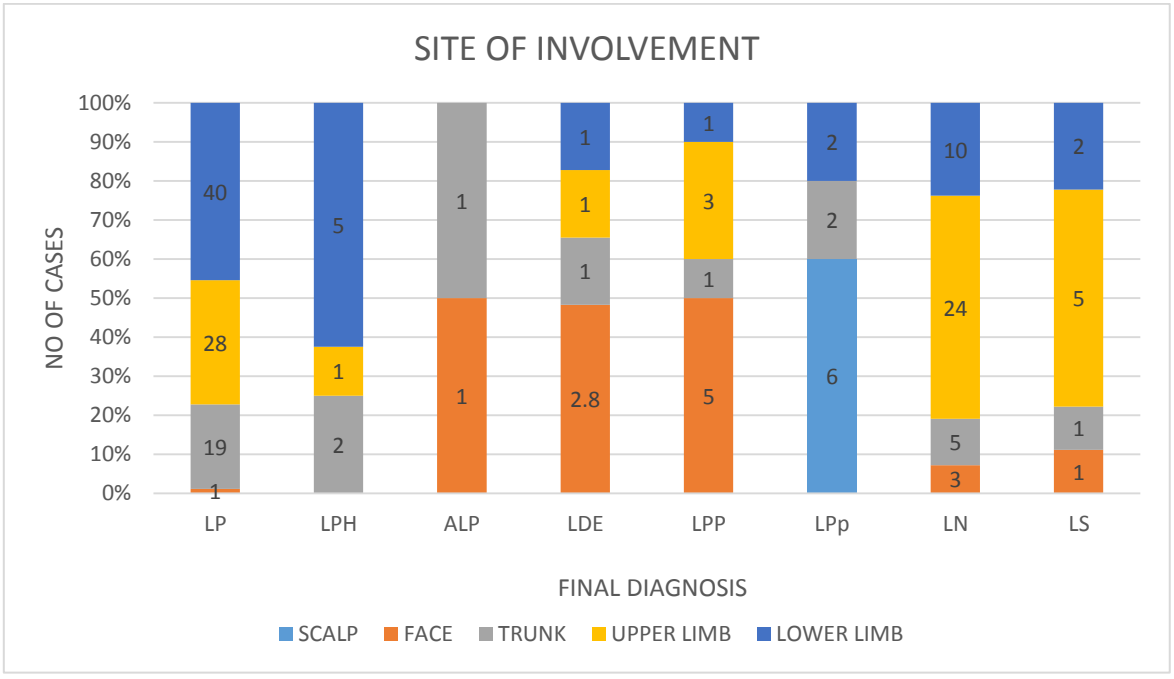

Figure 14: Bar diagram showing site involved according to final diagnosis

On dermoscopic examination

Wickham's striae: It was seen in 91.1% cases of Lichen planus (Figure 15) with predominantly radial streaming pattern (63%) (Figure 26), reticulate pattern (41.4%) (Figure 27) (Figure 16). The colour of WS seen was white (82.9%) (Figure 35), bluish white (21.9%) (Figure 36) and yellow (7.3%) as mentioned in table 9a and Figure 17.

Background: Violet colour background was seen in Lichen planus (75.5%) (Figure 38), Actinic LP (50%) (Figure 45), Lichen planus pigmentosus (20%). Brown colour background was seen in Lichen planus pigmentosus (100%), Lichen planopilaris (42.8%) and Lichen planus (28.8%) (Figure 37). Pink colour background was seen in Actinic lichen planus (100%) and Lichen planus (17.7%) as in Table 9b, Figure 18.

Pigment pattern: Greyish blue globules was seen in Lichen planus hypertrophicus (100%), Lichen planopilaris (71.4%) and Lichen planus (17.7%). Brown globules were noted in Lichen planus pigmentous (100%) (Figure 47), Lichenoid drug eruption (100%), Lichen planus hypertrophicus (80%) and Lichen planus (60%). No pigment pattern was seen in lichen nitidus (100%) and lichen striatus (100%) as mentioned in table 9b, Figure 19.

Arrangement of pigment: Diffuse type of pigmentation was seen in Lichen planus (100%), Lichen planus hypertrophicus (100%) and Lichen planus pigmentosus (100%) (Figure 47). Perifollicular type of pigmentation was seen in Lichen planus pigmentosus (100%) (Figure 47) and Lichen planopilaris (85.7%) (Table 9b and Figure 20).

Vascular pattern: Red dots were seen in Lichen planus (11.1%). Red globules were seen in Lichen planus (40%) (Figure 39) and Lichen planus hypertrophicus (20%). Red linear vessels were seen in Lichen planus (13.3%) (Figure 41) and peripheral homogenous erythema (Figure 40) was seen in Lichen planus (6.6%) and Lichen planopilaris (14.2%). Vascular pattern was absent in Lichen planus (46.6%), Lichen planus hypertrophicus (80%), Lichen planus pigmentosus (100%), Lichen planopilaris (85.7%), Lichen nitidus (100%) and Lichen striatus (100%) (Table 9c, Figure 21).

Other findings:

White globules were seen in Lichen nitidus (100%) (Figure 51) and Lichen striatus (100%) which were coalescing (Figure 53).

In Lichen planus hypertrophicus, pearly white striations (80%), yellow structures (80%) and comedo like openings (80%) were seen.

In Lichen planopilaris, empty follicles (100%), perifollicular scales and casts (100%) and structureless areas (57.1%) were seen (Figure 49).

Table 9a: Dermoscopic findings of lichenoid dermatoses

	LP (n= 45)		LPH (n=5)		ALP (n=2)		LDE (n=1)		LPP (n=5)		LPp (n=7)		LN (n=27)		LS (n=8)	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
WICKHAM'S STRIAE																
PRESENT	41	91.1	-	-	-	-	-	-	-	-	-	-	-	-	-	-
ABSENT	4	8.9	5	100	2	100	1	100	5	100	7	100	27	100	8	100
MORPHOLOGY OF WS																
RADIAL STREAMING	26	63	-	-	-	-	-	-	-	-	-	-	-	-	-	-
RETICULATE	17	41.4	-	-	-	-	-	-	-	-	-	-	-	-	-	-
VEIL LIKE	7	17	-	-	-	-	-	-	-	-	-	-	-	-	-	-
LINEAR	6	14.6	-	-	-	-	-	-	-	-	-	-	-	-	-	-
CIRCULAR	3	7.3	-	-	-	-	-	-	-	-	-	-	-	-	-	-
LEAF LIKE	1	2.4	-	-	-	-	-	-	-	-	-	-	-	-	-	-
CIRCULAR RETICULATE	2	4.8	-	-	-	-	-	-	-	-	-	-	-	-	-	-
COLOUR OF WS																
WHITE	34	82.9	-	-	-	-	-	-	-	-	-	-	-	-	-	-
BLUISH WHITE	9	21.9	-	-	-	-	-	-	-	-	-	-	-	-	-	-
YELLOW	3	7.3	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Table 9b: Dermoscopic findings of lichenoid dermatoses

	LP (n= 45)		LPH (n=5)		ALP (n=2)		LDE (n=1)		LPP (n=5)		LPp (n=7)		LN (n=27)		LS (n=8)	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
BACKGROUND																
VIOLET	34	75.5	-	-	1	50	-	-	1	20	1	14.2	-	-	-	-
BROWN	13	28.8	-	-	1	50	1	100	5	100	3	42.8	-	-	-	-
PINK	8	17.7	-	-	2	100	1	100	-	-	1	14.2	-	-	-	-
NO BACK-GROUND	3	6.6	-	-	-	-	-	-	-	-	2	28.5	27	100	8	100
PIGMENT PATTERN																
BLACKISH BLUE GLOBULES	5	11.1	-	-	-	-	-	-	1	20	-	-	-	-	-	-
GREYISH BLUE GLOBULES	8	17.7	5	100	-	-	-	-	1	20	5	71.4	-	-	-	-
BROWN GLOBULES	27	60	4	80	1	50	1	100	5	100	1	14.2	-	-	-	-
ABSENT	9	20	-	-	1	50	-	-	-	-	1	14.2	27	100	8	100
ARRANGE--MENT OF PIGMENT PATTERN																
DIFFUSE	36	100	5	100	1	50	1	100	5	100	-	-	-	-	-	-
PERI-FOLLICULAR	-	-	-	-	-	-	-	-	5	100	6	85.7	-	-	-	-

Table 9c: Dermoscopic findings of lichenoid dermatoses

	LP (n= 45)		LPH (n=5)		ALP (n=2)		LDE (n=1)		LPP (n=5)		LPp (n=7)		LN (n=27)		LS (n=8)	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
VASCULAR PATTERN																
RED DOTS	5	11.1	-	-	1	50	-	-	-	-	-	-	-	-	-	-
RED GLOBULES	18	40	1	20	-	-	1	100	-	-	-	-	-	-	-	-
RED LINEAR	6	13.3	-	-	1	50	-	-	-	-	-	-	-	-	-	-
PERIPHERAL HOMOGENOUS	3	6.6	-	-	-	-	-	-	-	-	1	14.2	-	-	-	-
ABSENT	21	46.6	4	80	-	-	-	-	5	100	6	85.7	27	100	8	100
OTHERS																
-WHITE GLOBULES	-	-	1	20	-	-	-	-	-	-	-	-	27	100	8	100
- PEARLY WHITE WITH STRIATIONS	-	-	4	80	-	-	-	-	-	-	-	-	-	-	-	-
- YELLOW STRUCTURES	-	-	4	80	-	-	-	-	-	-	-	-	-	-	-	-
- COMEDO LIKEOPENINGS	4	8.8	4	80	-	-	-	-	-	-	-	-	-	-	-	-
- MILIUM LIKE CYST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
- EMPTY FOLLICLE	-	-	-	-	-	-	-	-	-	-	7	100	-	-	-	-
-PERI-FOLLICULAR SCALES	-	-	-	-	-	-	-	-	-	-	7	100	-	-	-	-
-STRUCTURE-LESS AREAS	-	-	-	-	-	-	-	-	-	-	4	100	-	-	-	-

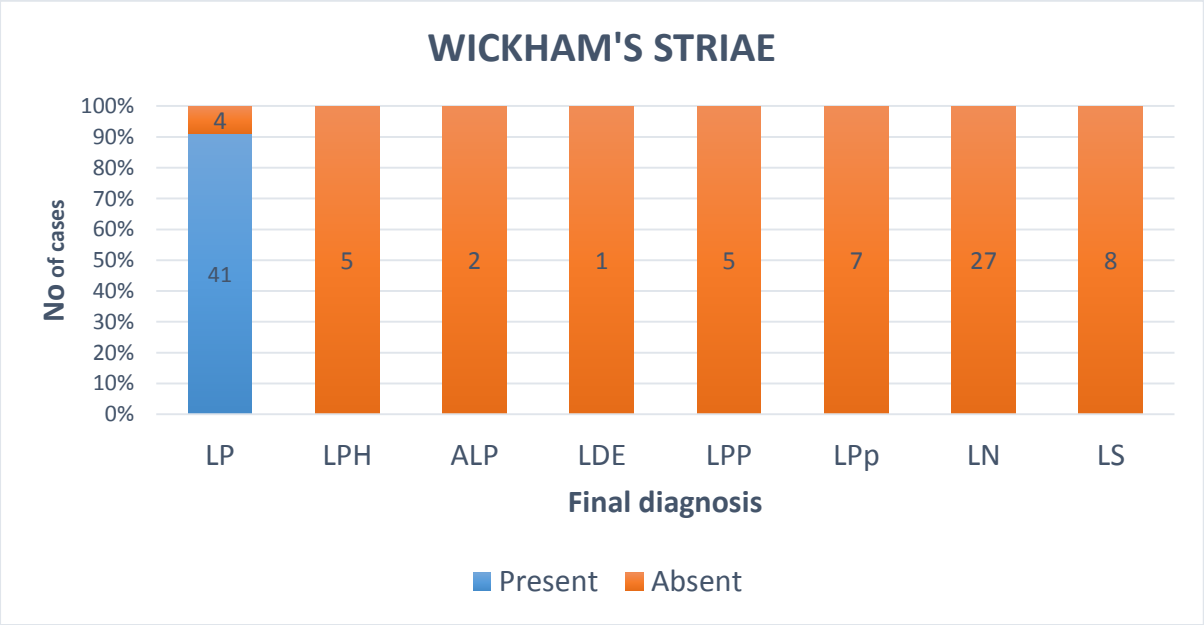

Figure 15: Bar diagram showing presence or absence of Wickham's striae in lichenoid dermatoses

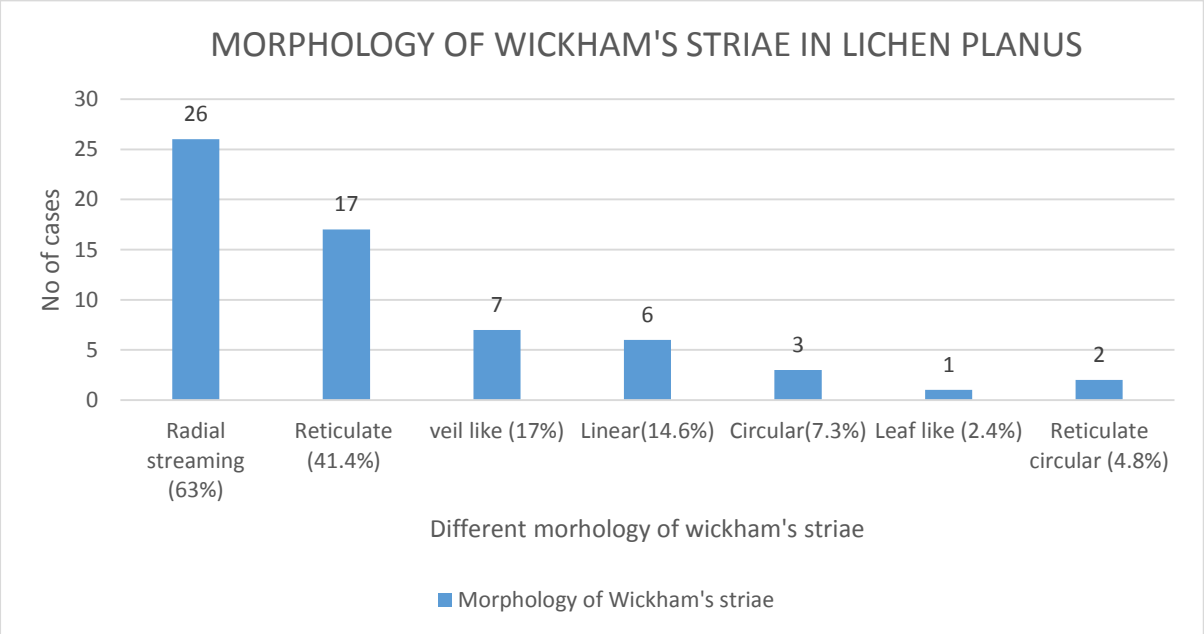

Figure 16: Bar diagram showing different morphology of Wickham's striae in lichen planus.

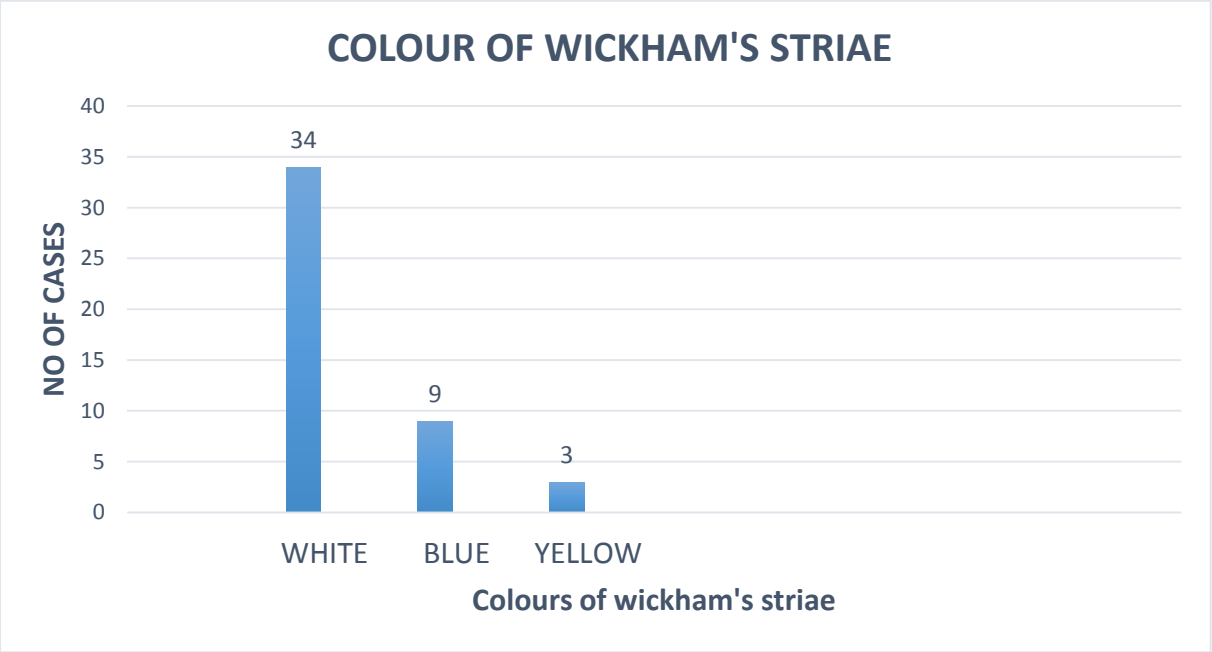

Figure 17: Bar diagram showing different colours of Wickham's striae in lichen planus.

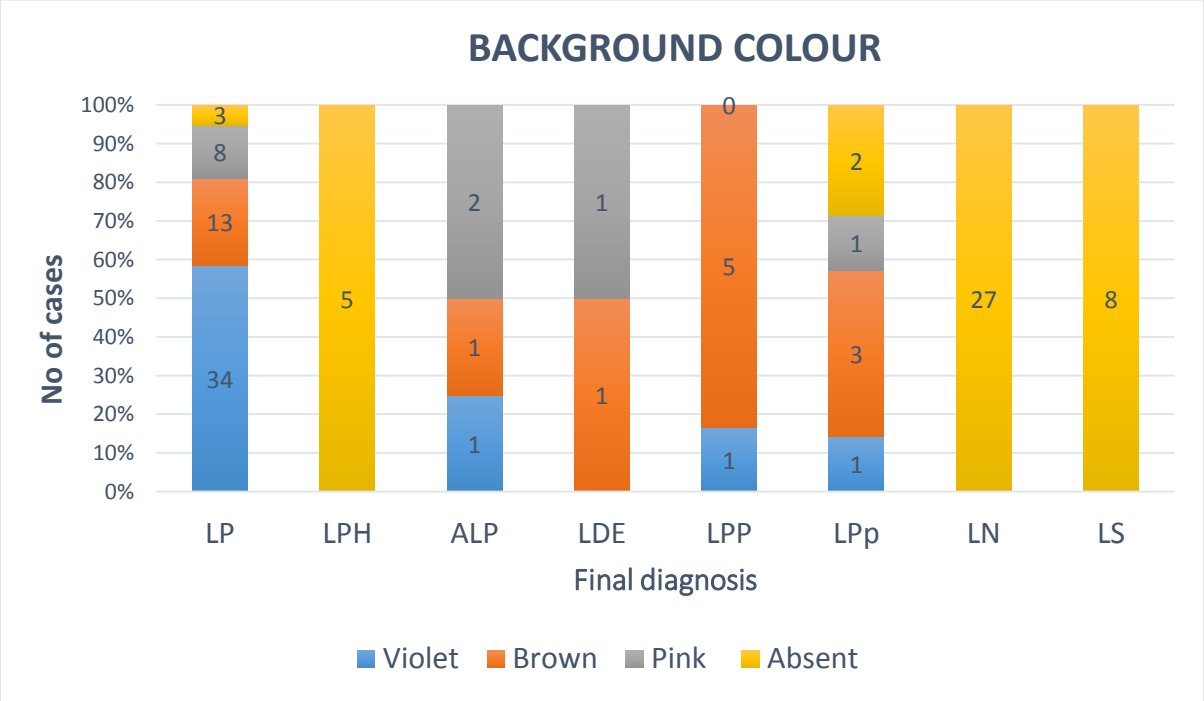

Figure 18: Bar diagram showing background colour in lichenoid dermatoses.

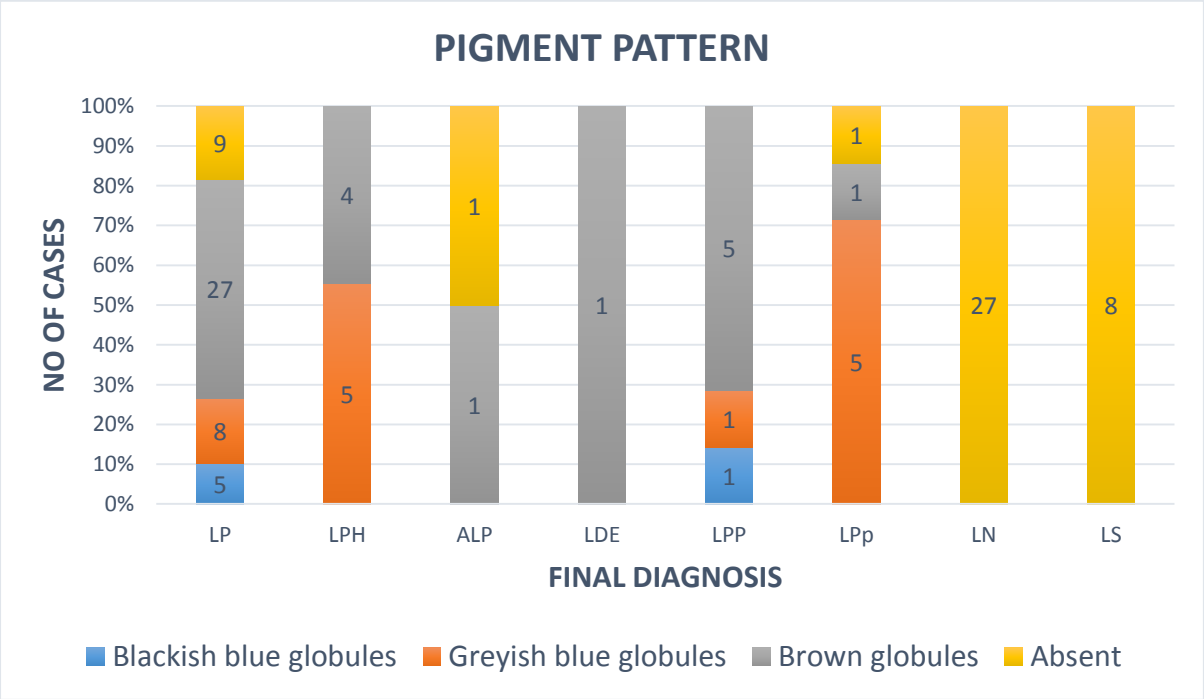

Figure 19: Bar diagram showing pigmentary patterns in lichenoid dermatoses

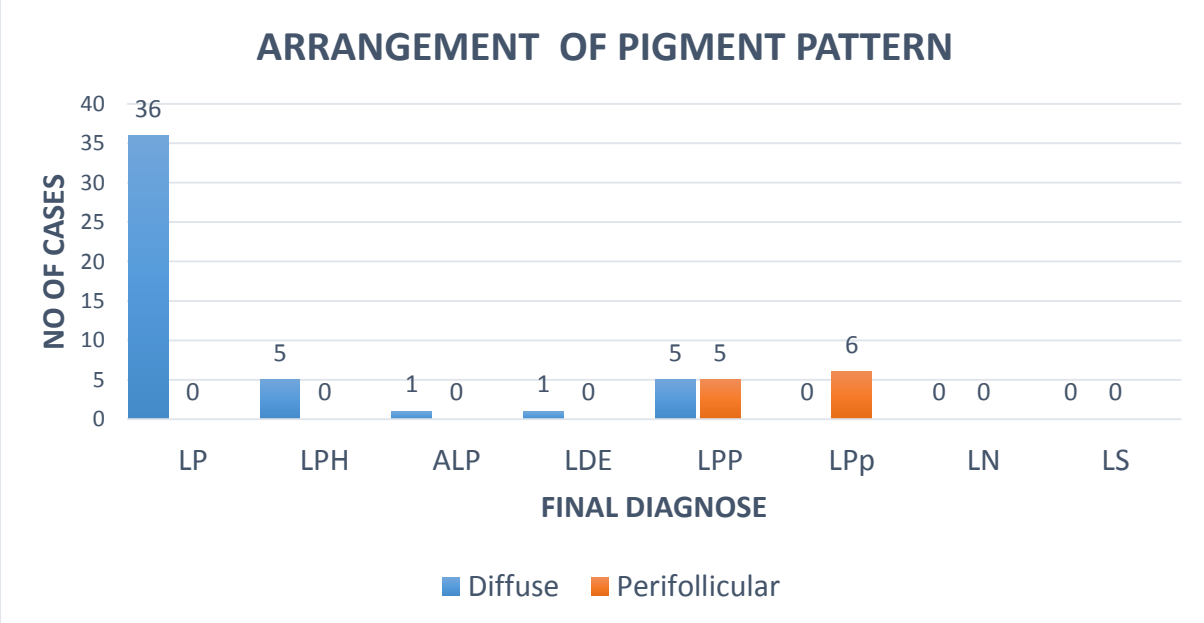

Figure 20: Bar diagram showing arrangement of pigment patterns in lichenoid dermatoses

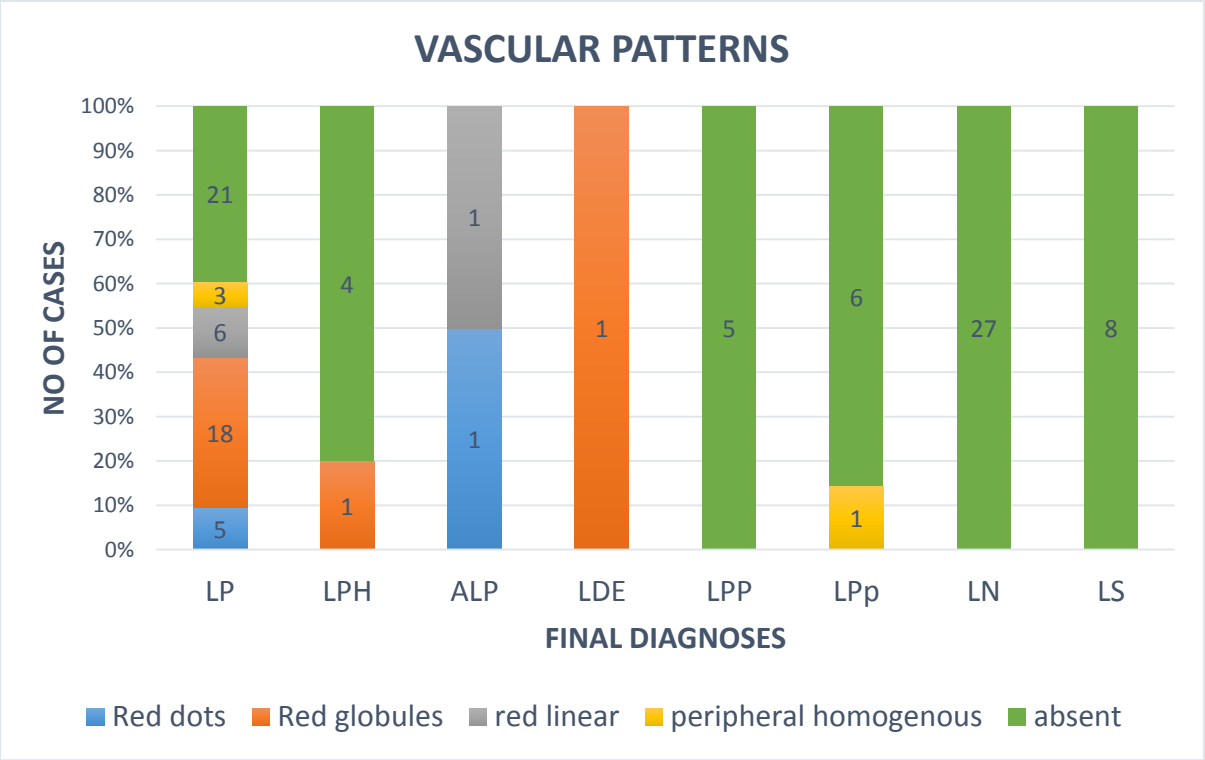

Figure 21: Bar diagram showing vacular patterns in lichenoid dermatoses

PICTURES

LICHEN PLANUS

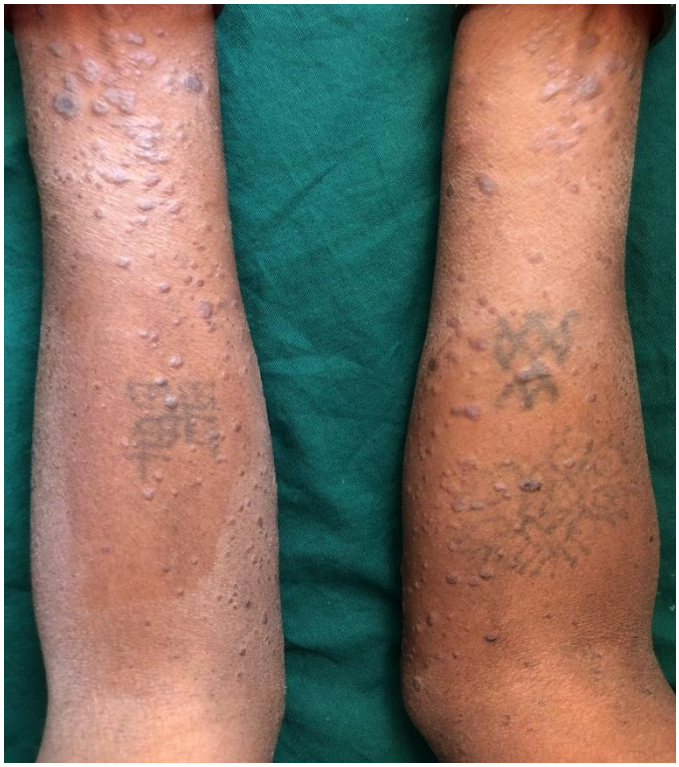

Figure 22: Violaceous papules on flexor aspect of forearm

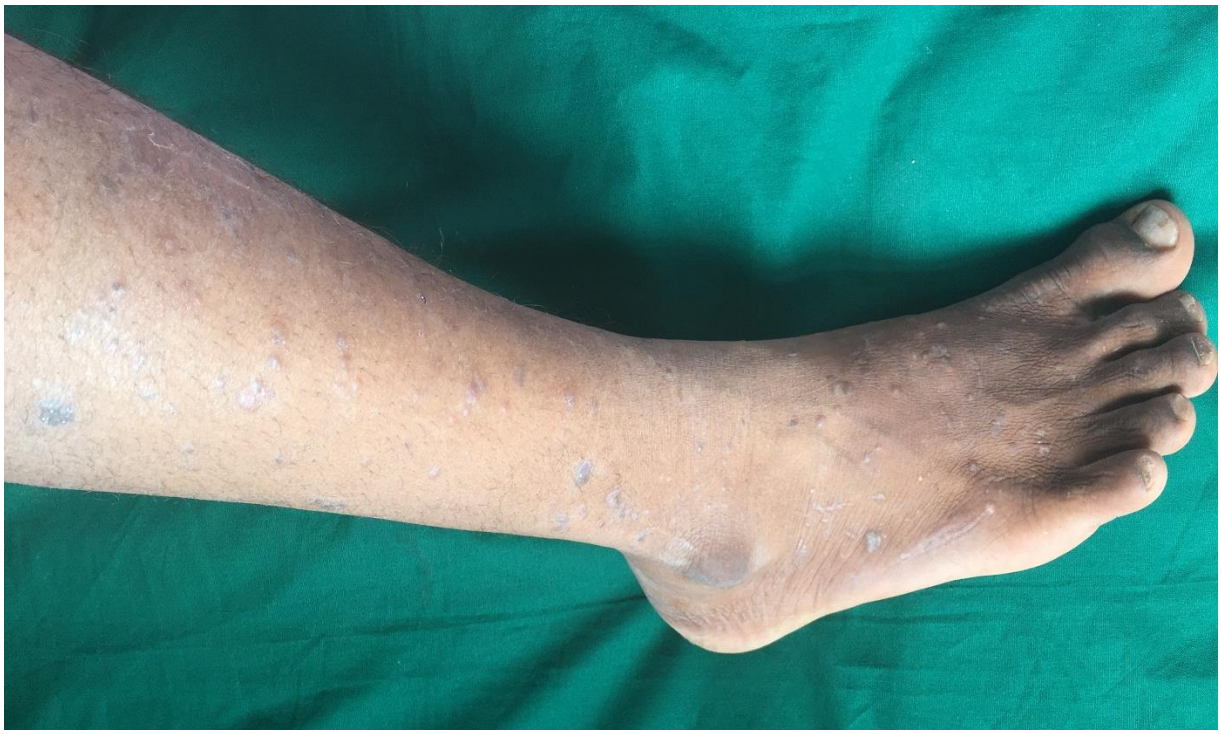

Figure 23: Violaceous papules on legs

Figure 24: violacoeus papules on back

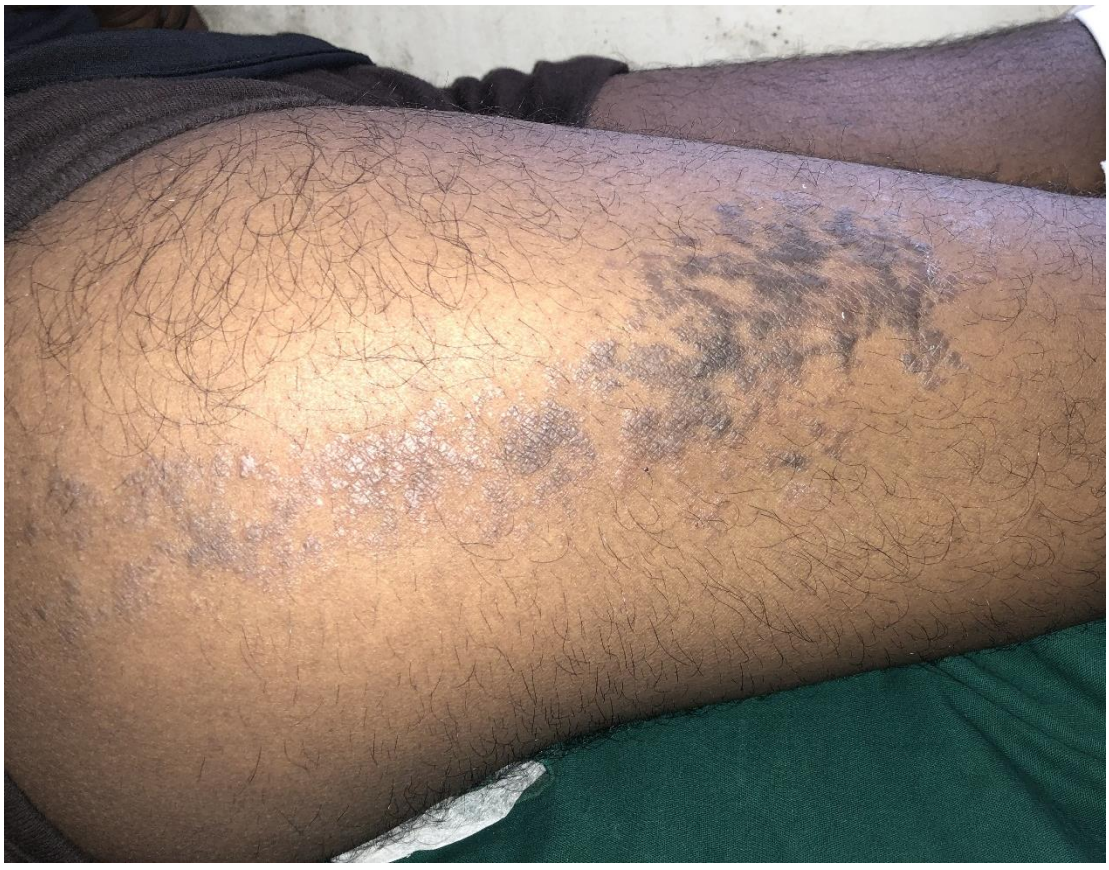

Figure 25: violacoeus papules on right thigh along the blashko's lines

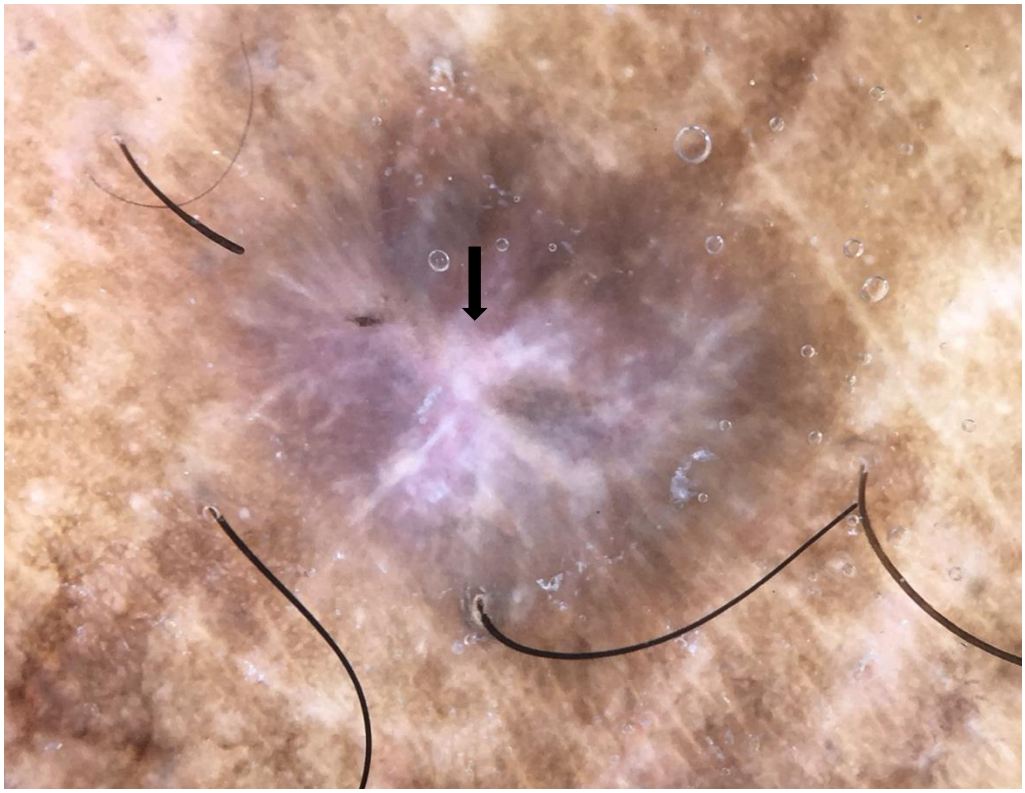

Figure 26: Radial streaming pattern of wickham's striae

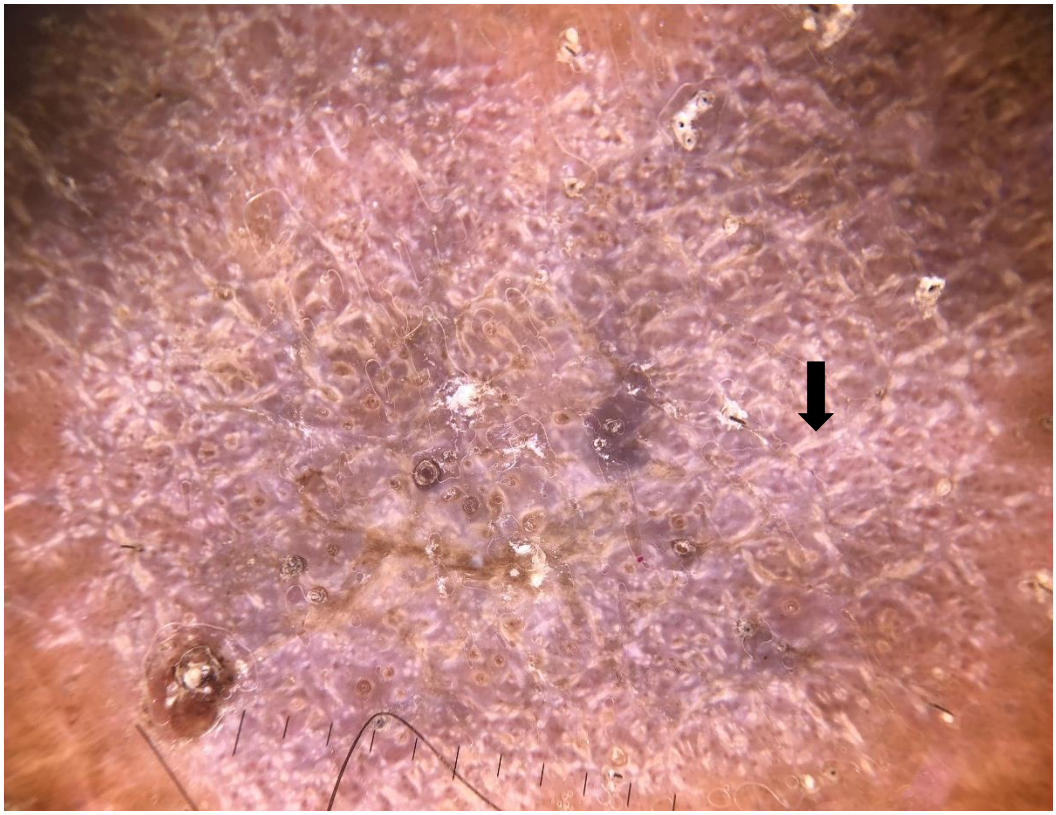

Figure 27: Reticulate pattern of wickham's striae

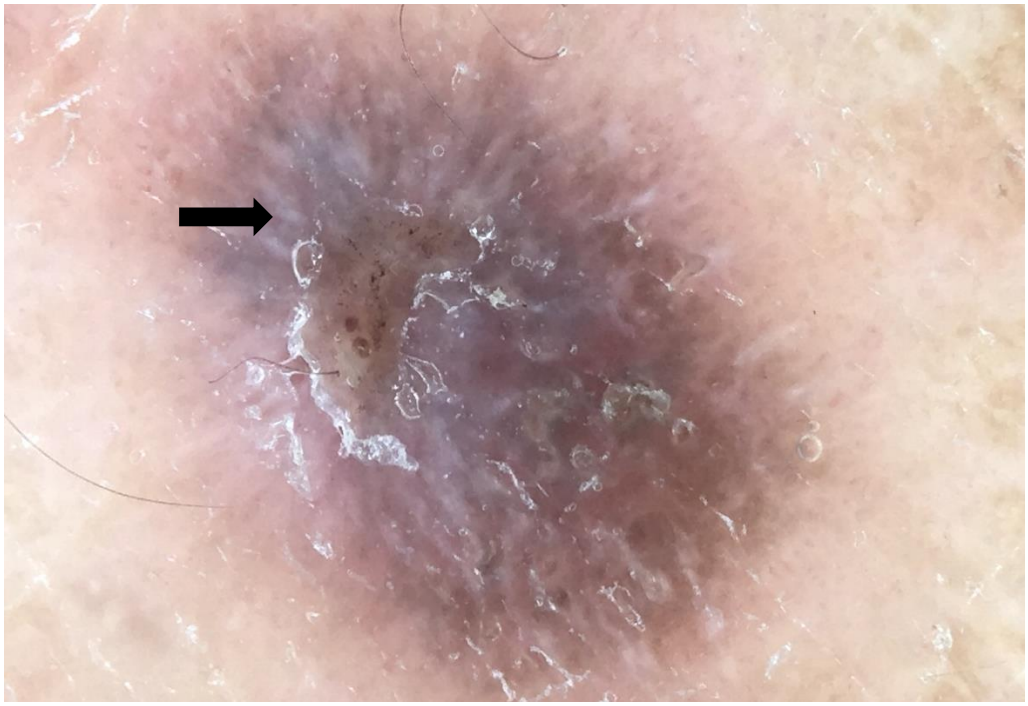

Figure 28: veil like pattern of wickham's striae

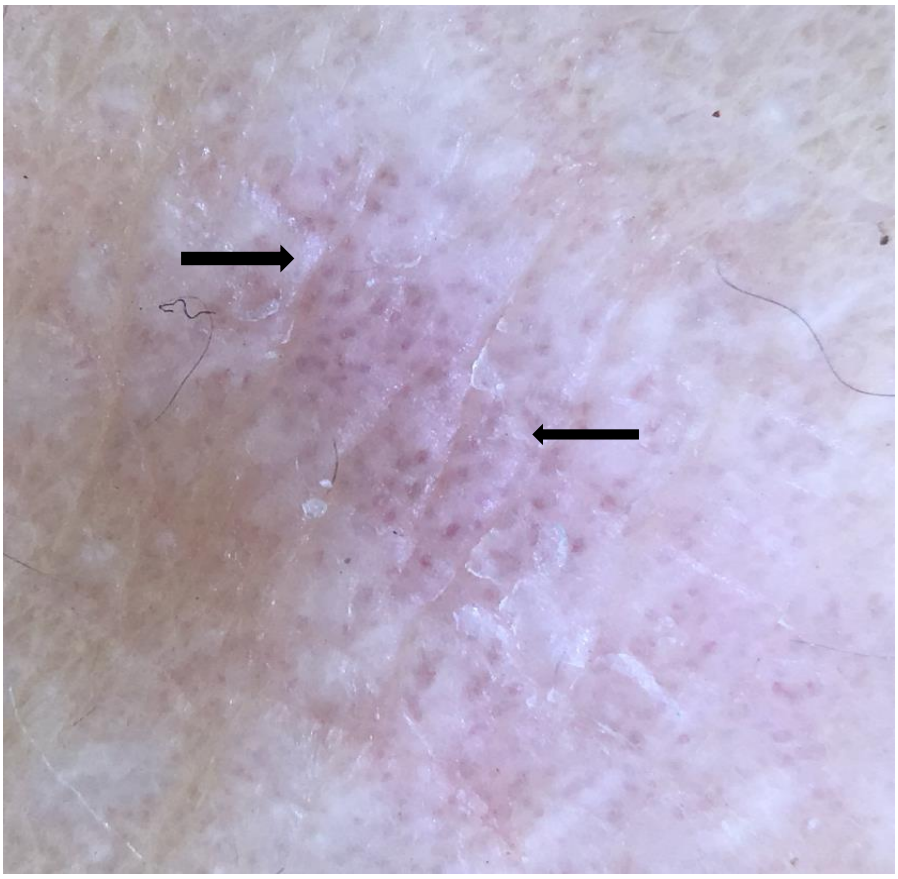

Figure 29: veil like pattern of wickham's striae

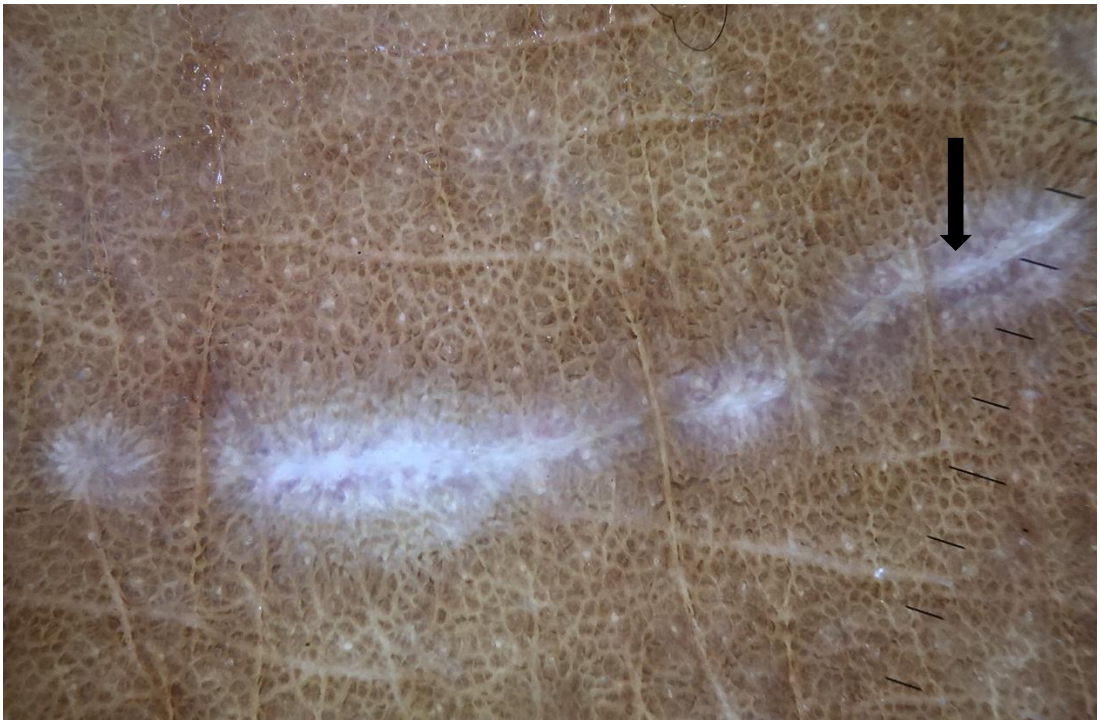

Figure 30: Linear pattern of wickham's striae

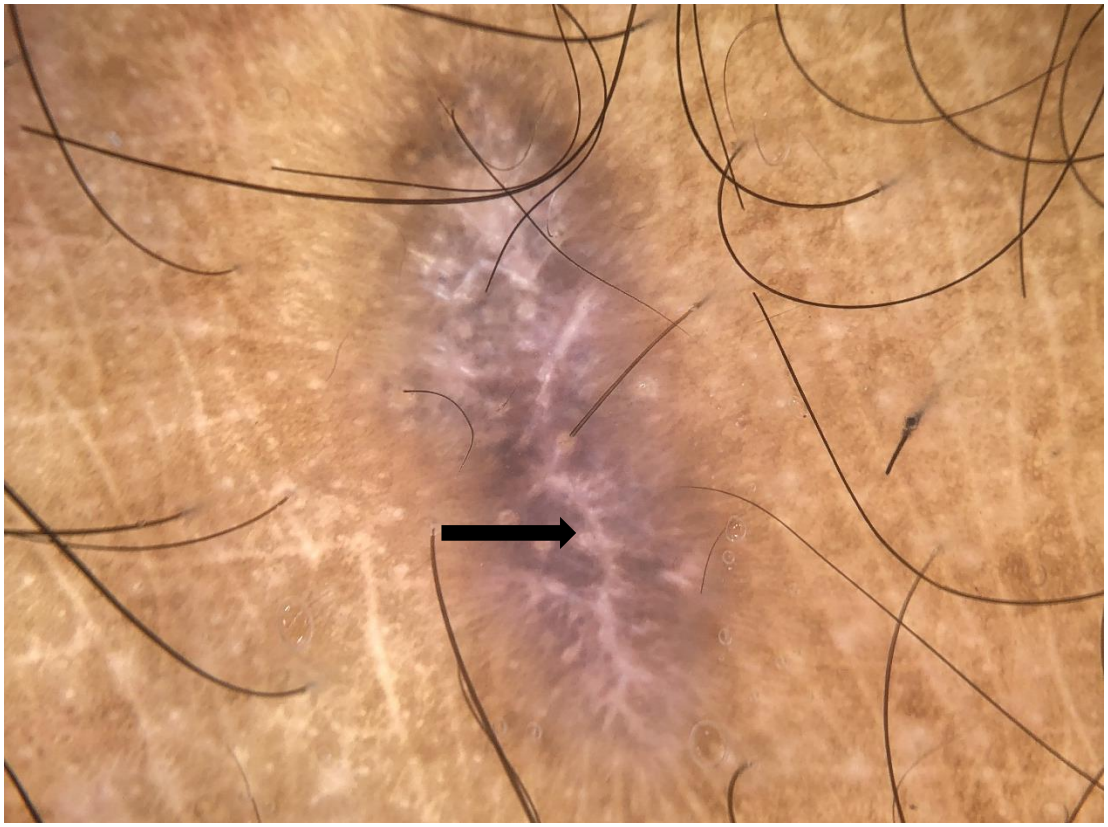

Figure 31: Linear pattern of wickham's striae

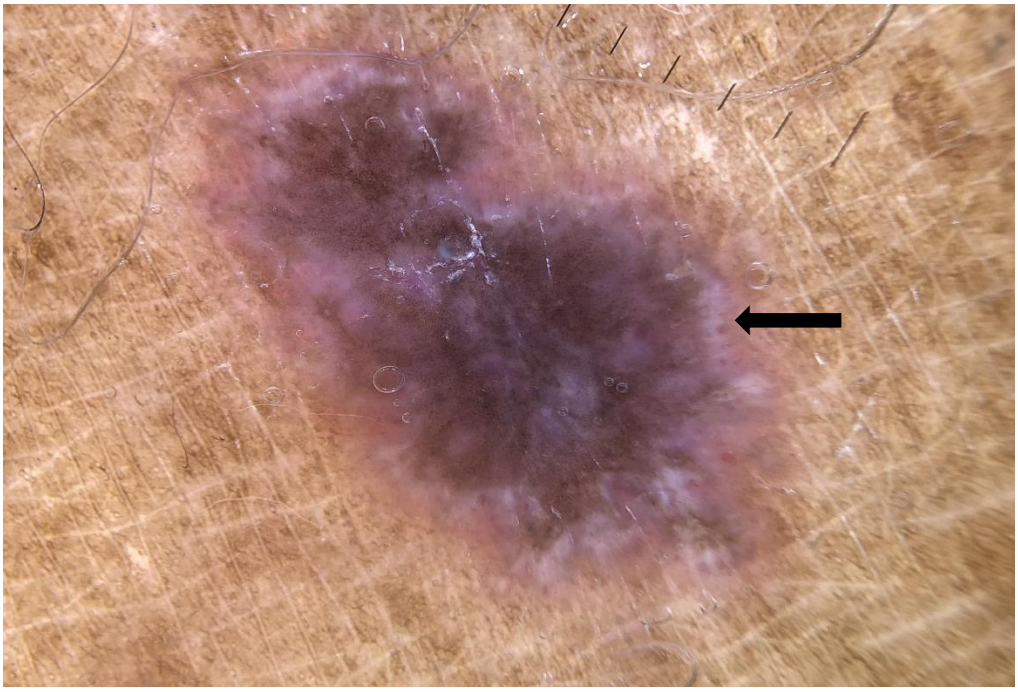

Figure 32: circular pattern of wickham's striae

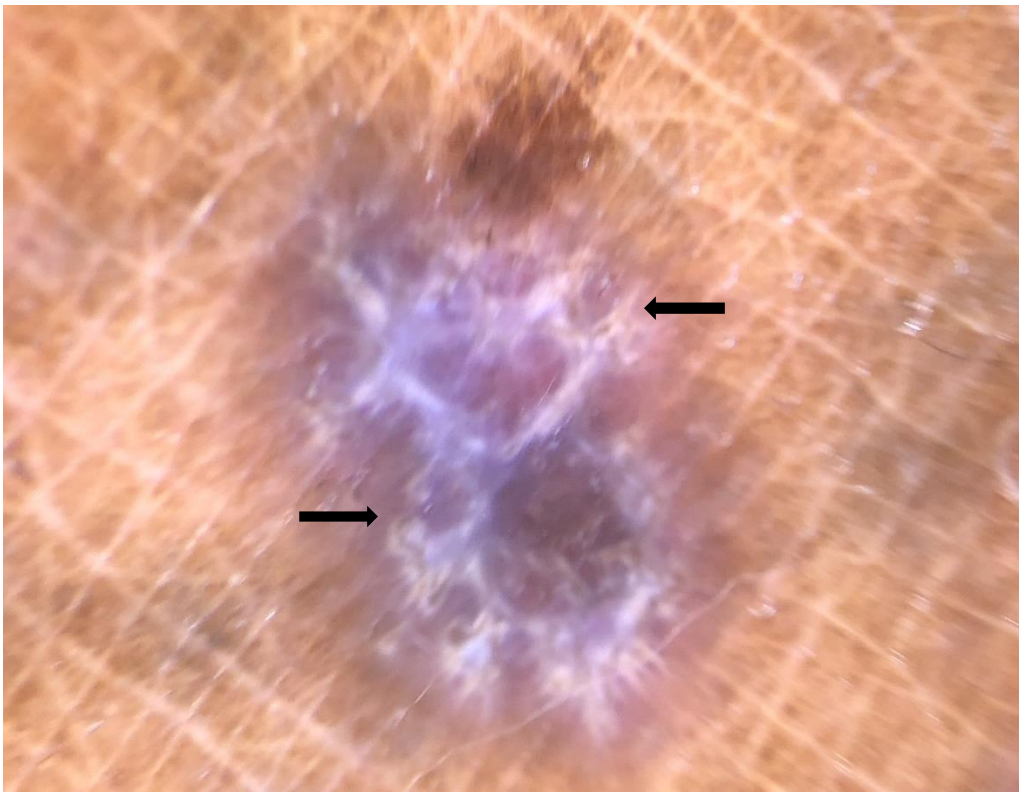

Figure 33: Circular Reticulate pattern of wickham's striae

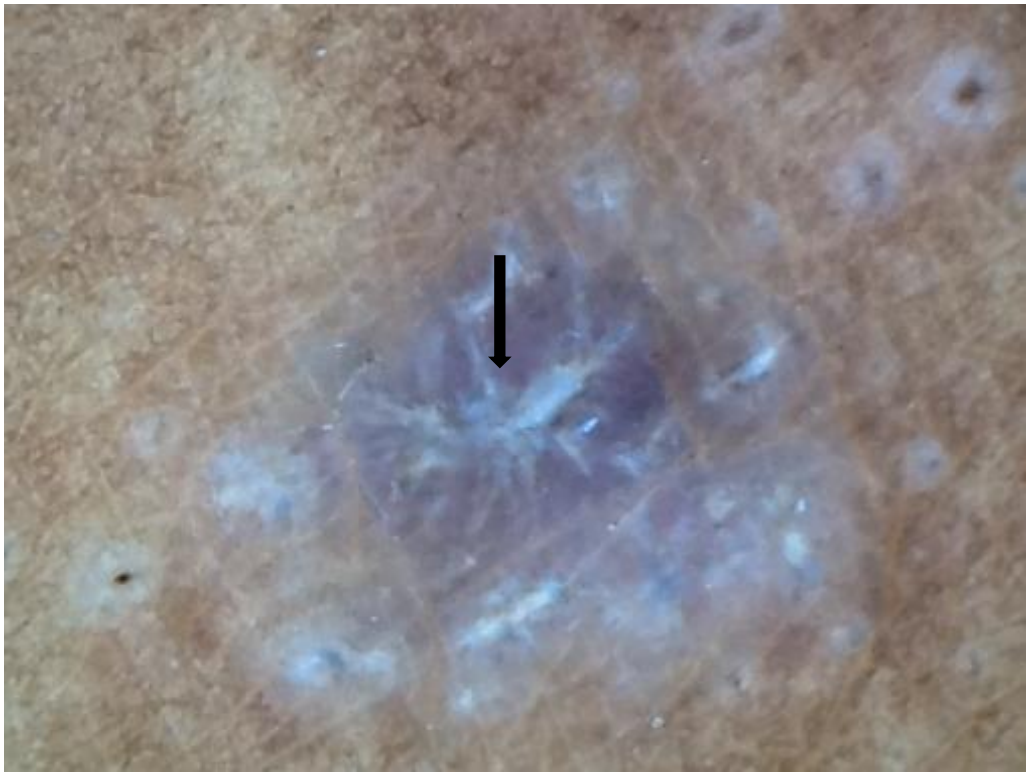

Figure 34: Leaf like pattern of wickham's striae

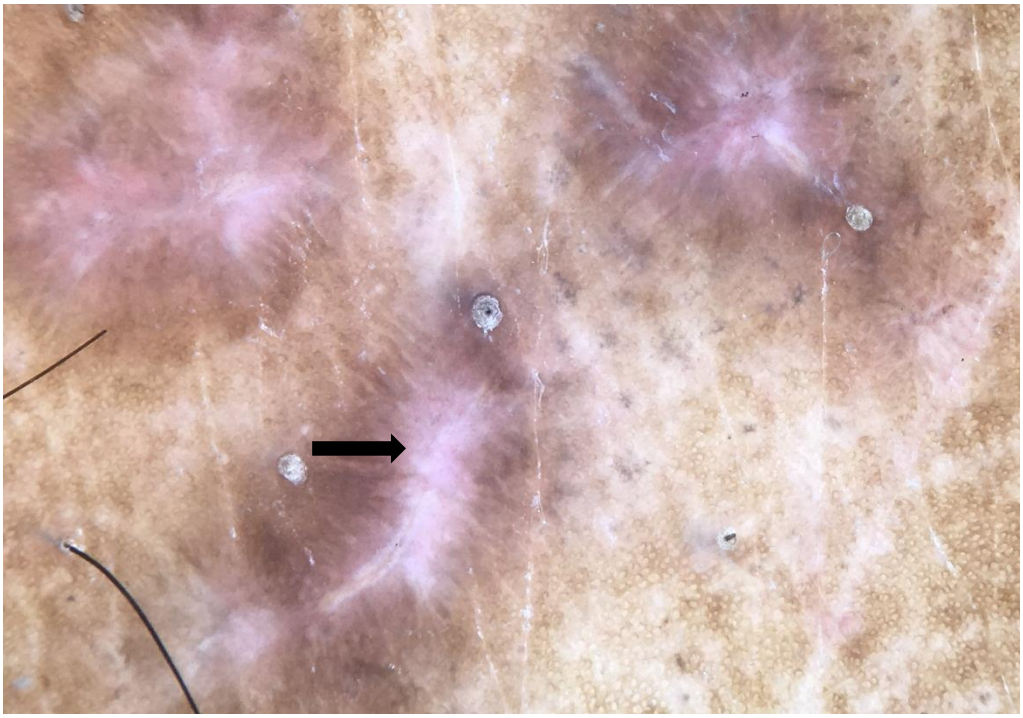

Figure 35: white colour of wickham's striae

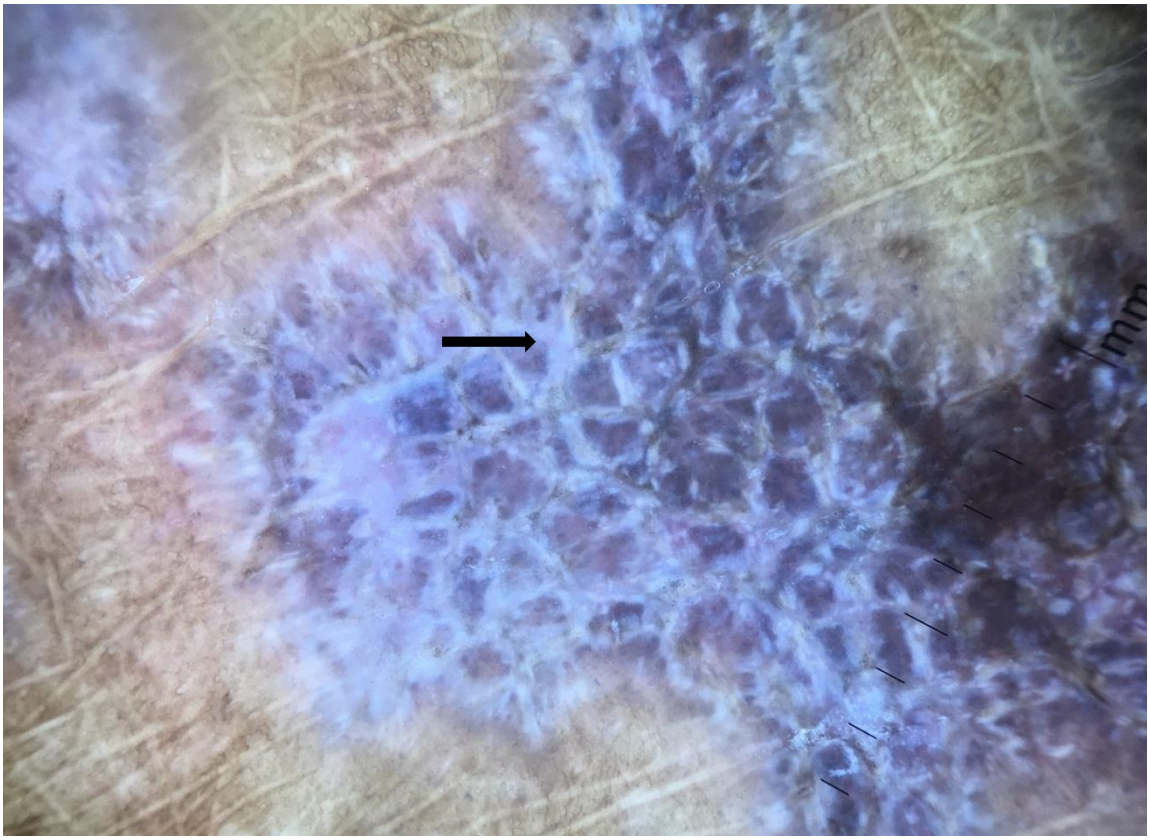

Figure 36: Bluish white type of wickham's striae

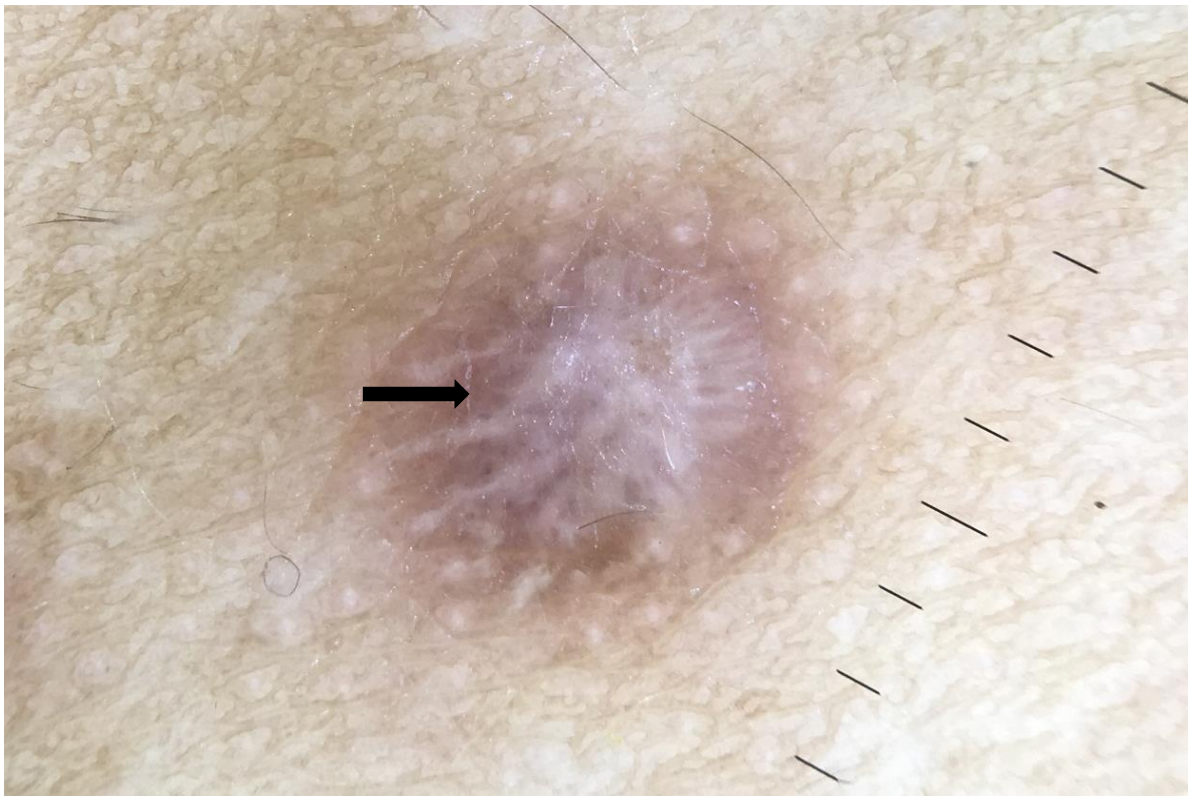

Figure 37: Brown background of lesion in lichen planus

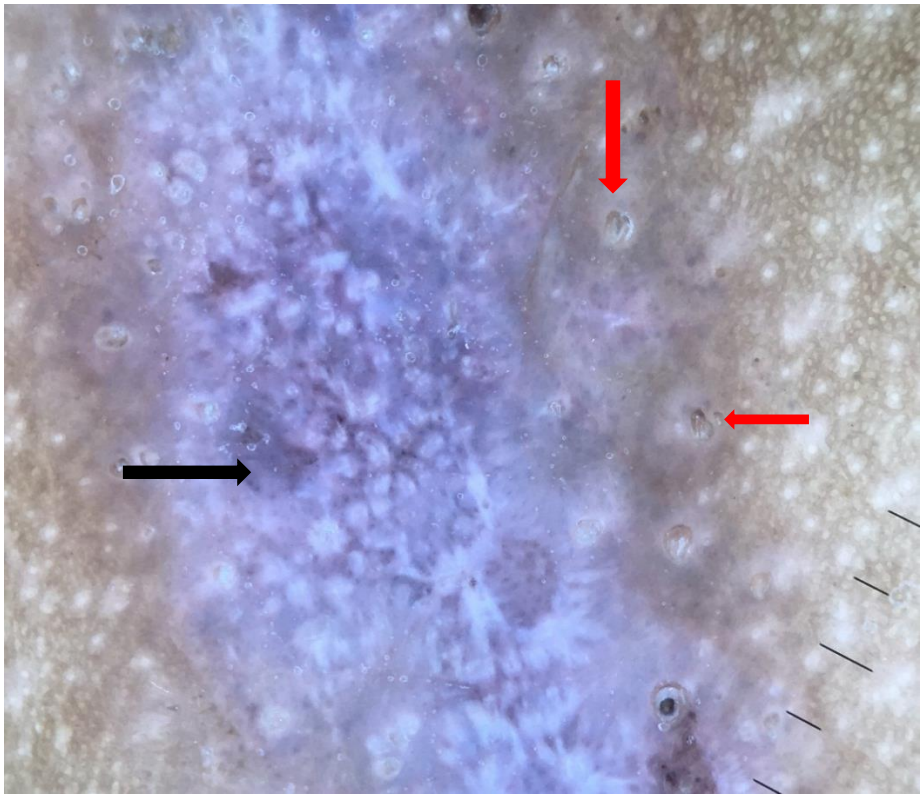

Figure 38: Violet back ground of lesion in lichen planus shown by black arrow and red arrow showing comedo like openings

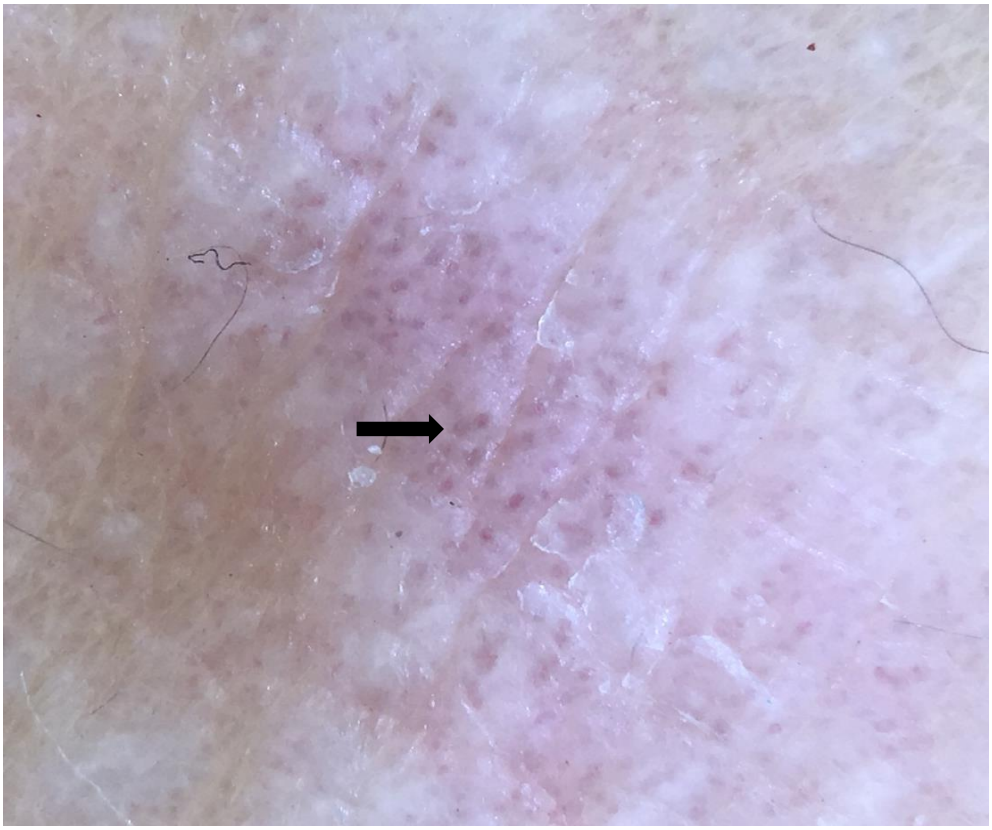

Figure 39: Red globules in lichen planus

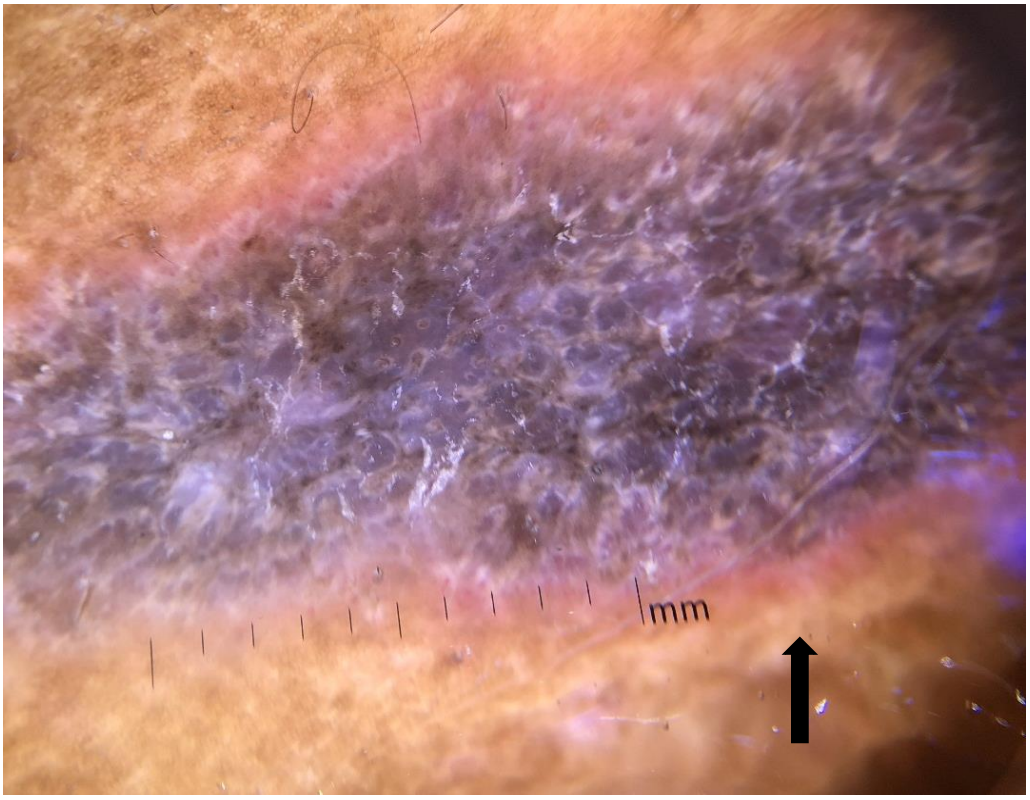

Figure 40: Peripheral homogenous erythema in lichen planus pointed by black arrow

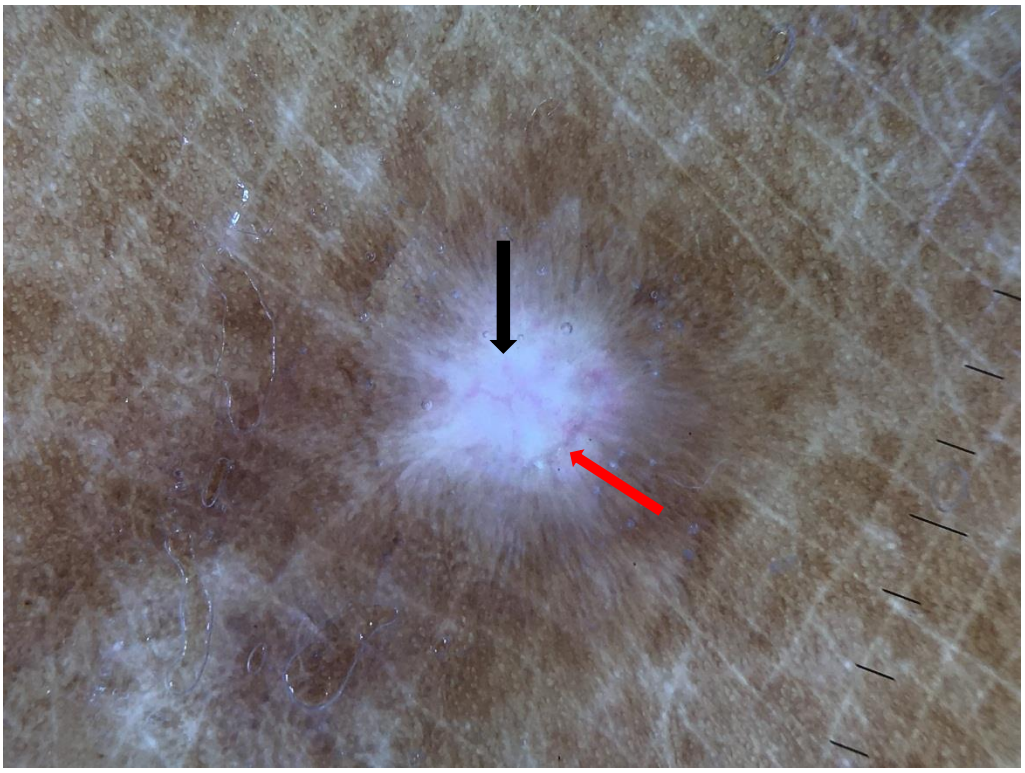

Figure 41: white Radial streaming type of wickham's striae (black arrow) and Red linear vessels (red arrow) in lichen planus

LICHEN PLANUS HYPERTROPHICUS

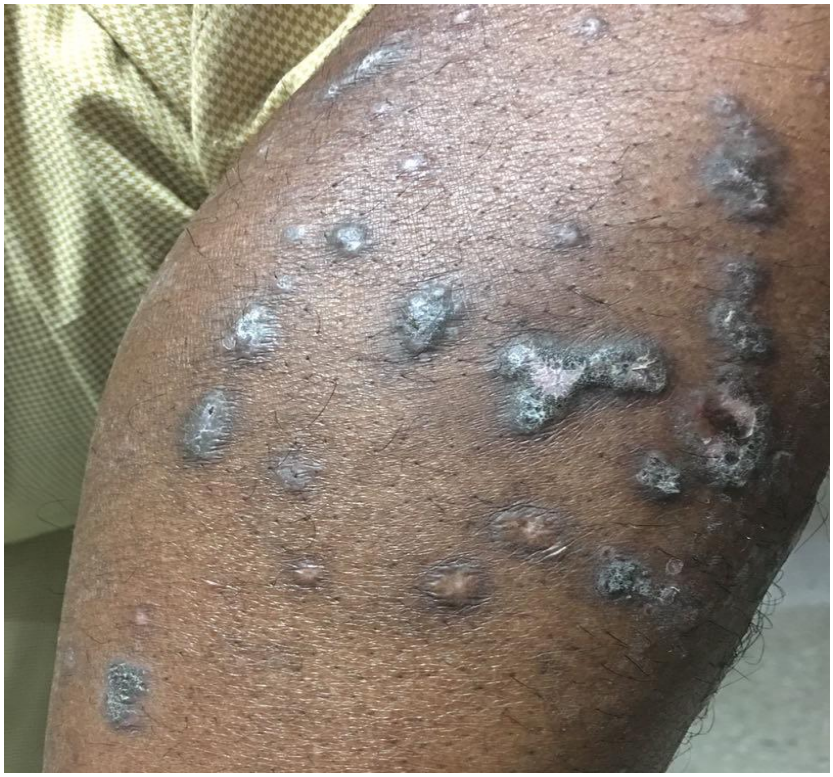

Figure 42: Multiple violaceous plaques present over right leg

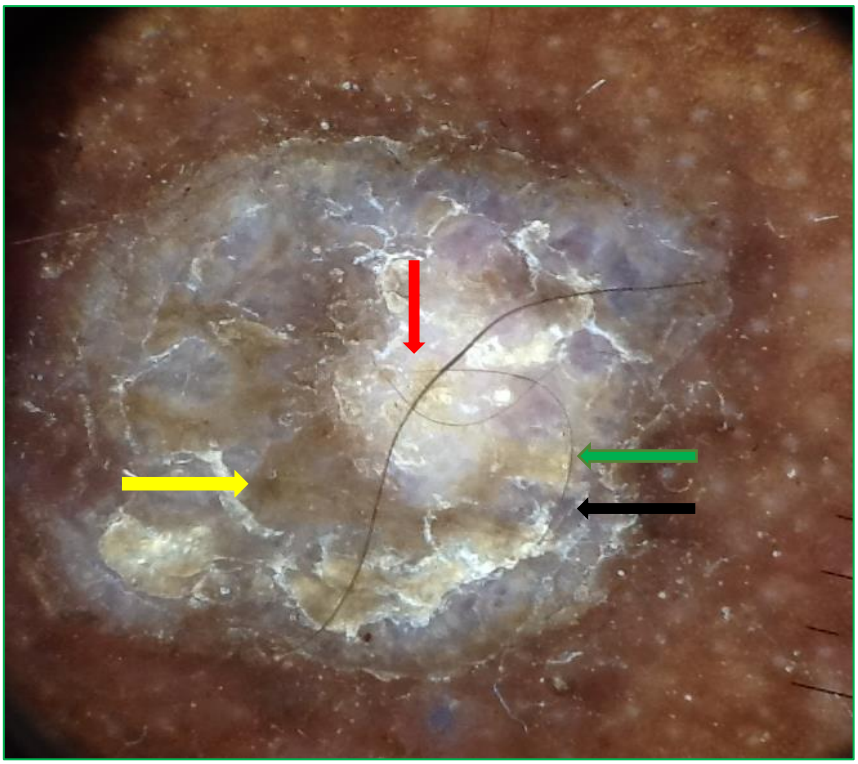

Fig 43: Lichen planus hypertrophicus. Red arrow denotes pearly white structureless areas, green arrow denotes yellow structure, yellow arrow denotes brown globules, black arrow denotes blackish blue globules.

LICHENOID DRUG ERUPTION

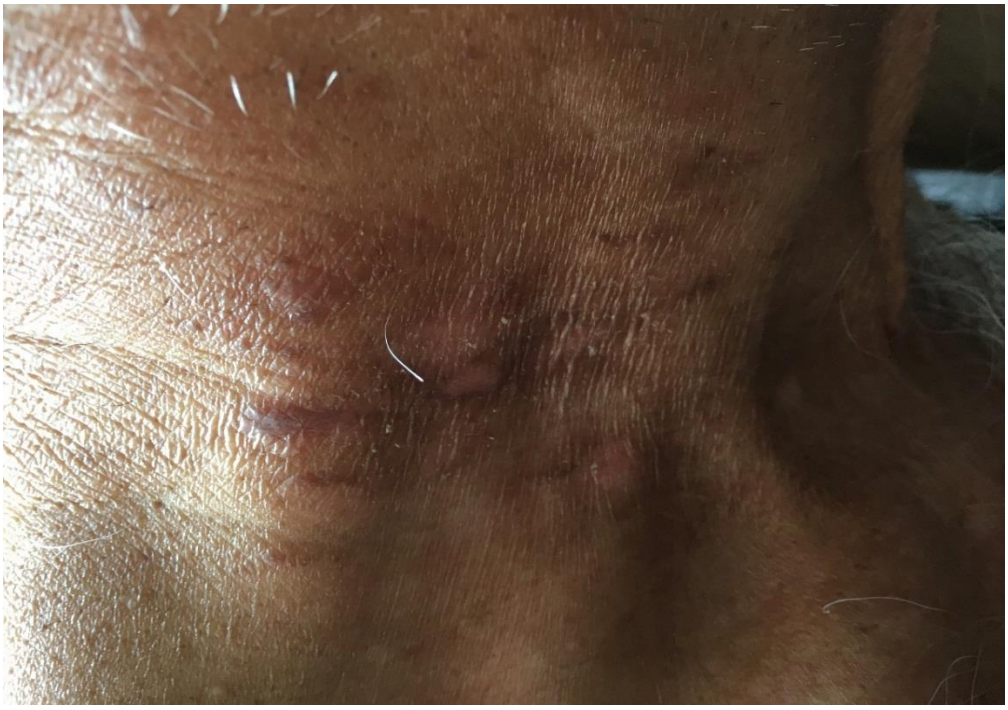

Figure 44 : Lichenoid drug eruption presenting with erythematous and violaceous papules

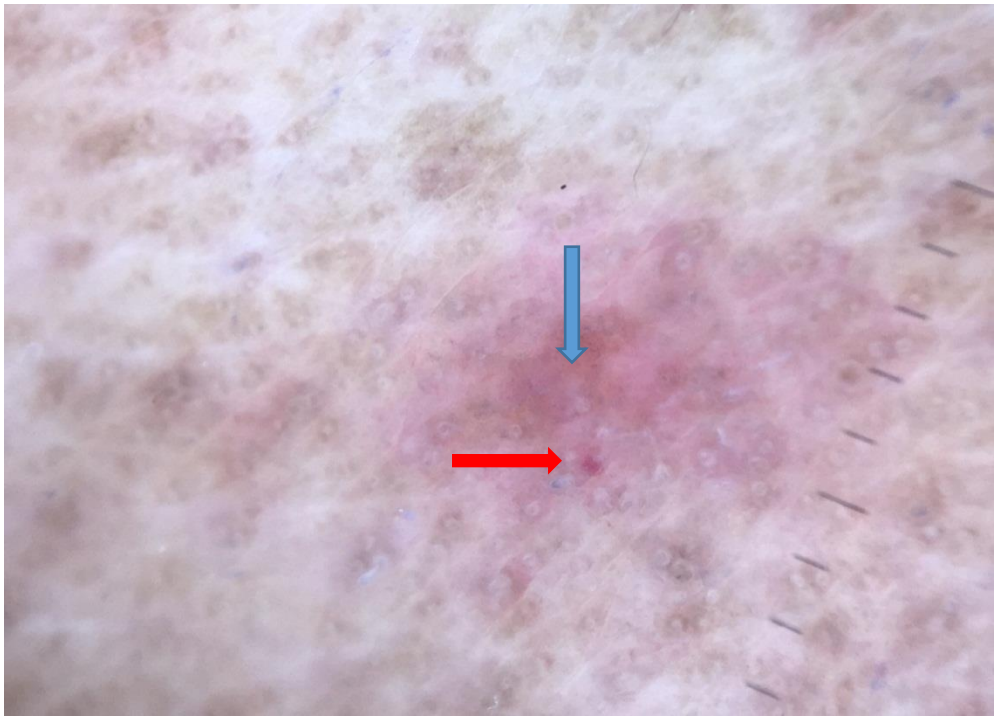

Figure 45: Lichenoid drug eruption. On dermoscopy, Absence of wickham's striae, blue arrow denotes pink background colour, red arrow denotes red globules.

LICHEN PLANUS PIGMENTOSUS

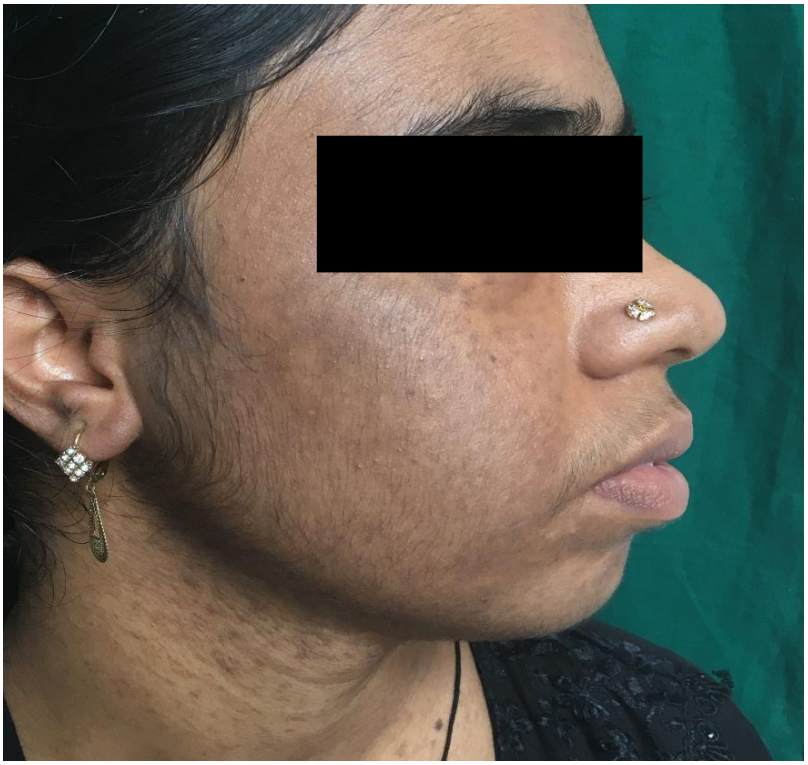

Figure 46: Slate grey pigmentation present over cheeks and neck

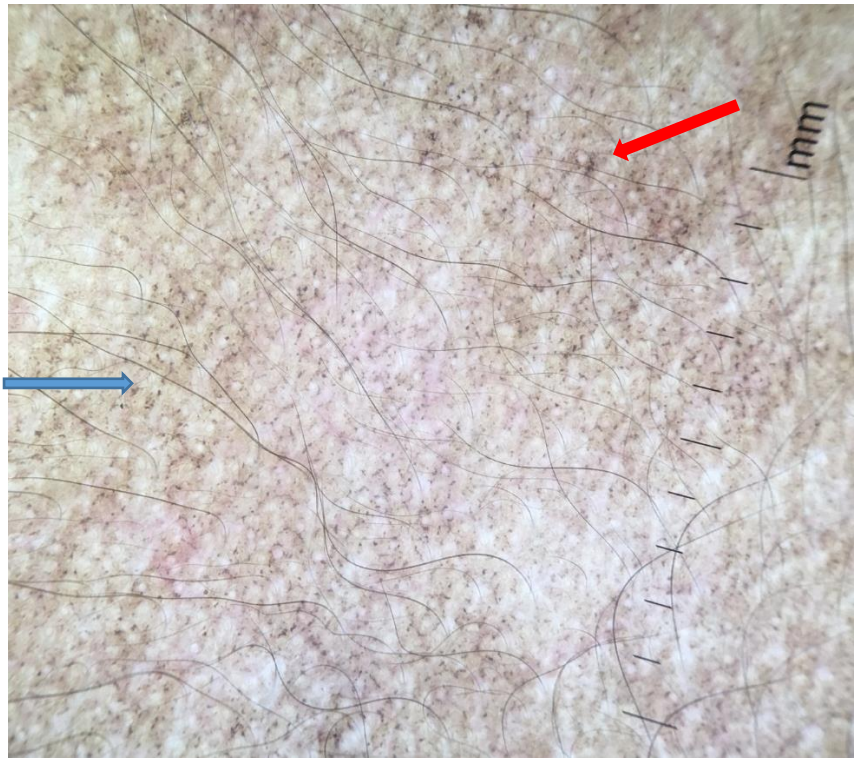

Fig 47: Lichen planus pigmentosus. On dermoscopy, Blue arrow denotes diffuse brown dots; red arrow denotes increased brown dots and globules around acrosyringeal openings

LICHEN PLANOPILARIS

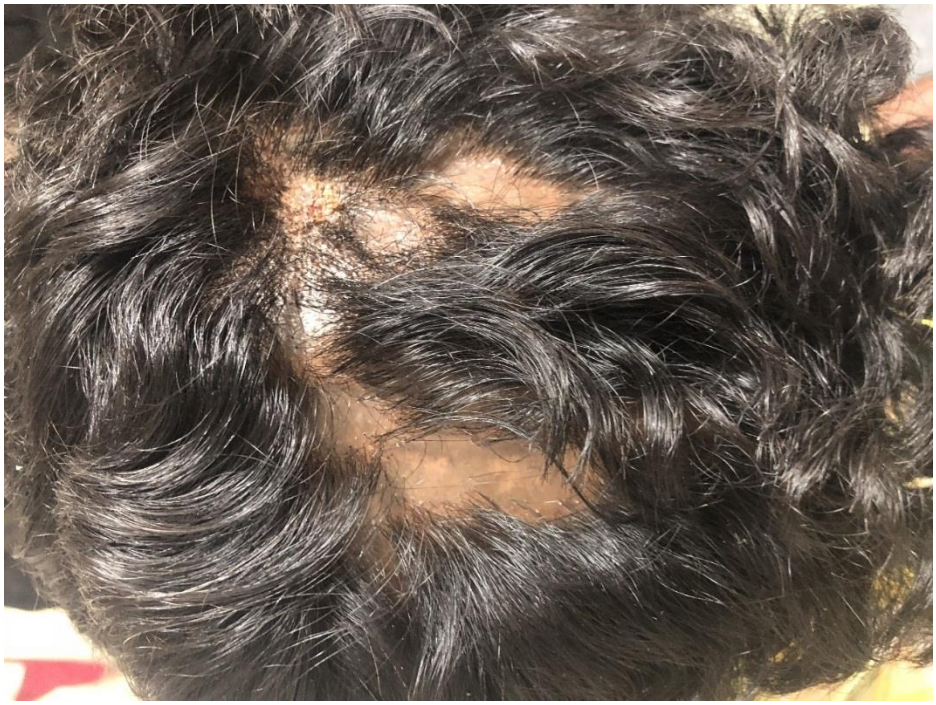

Figure 48: Patchy loss of hair with loss of hair follicles and surface appearing shiny

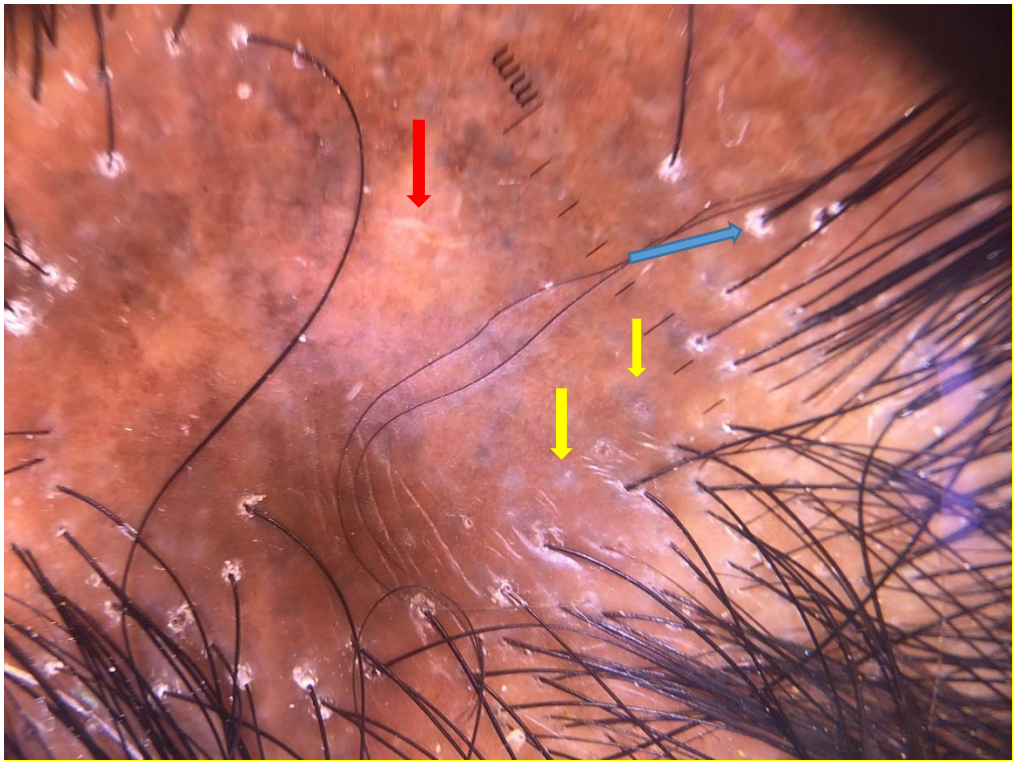

Fig 49: Lichen planopilaris. On dermoscopy, Blue arrow denotes perifollicular scaling, yellow arrow denotes greyish blue 'target pattern' pigmentation, Red arrow denotes structureless areas.

LICHEN NITIDUS

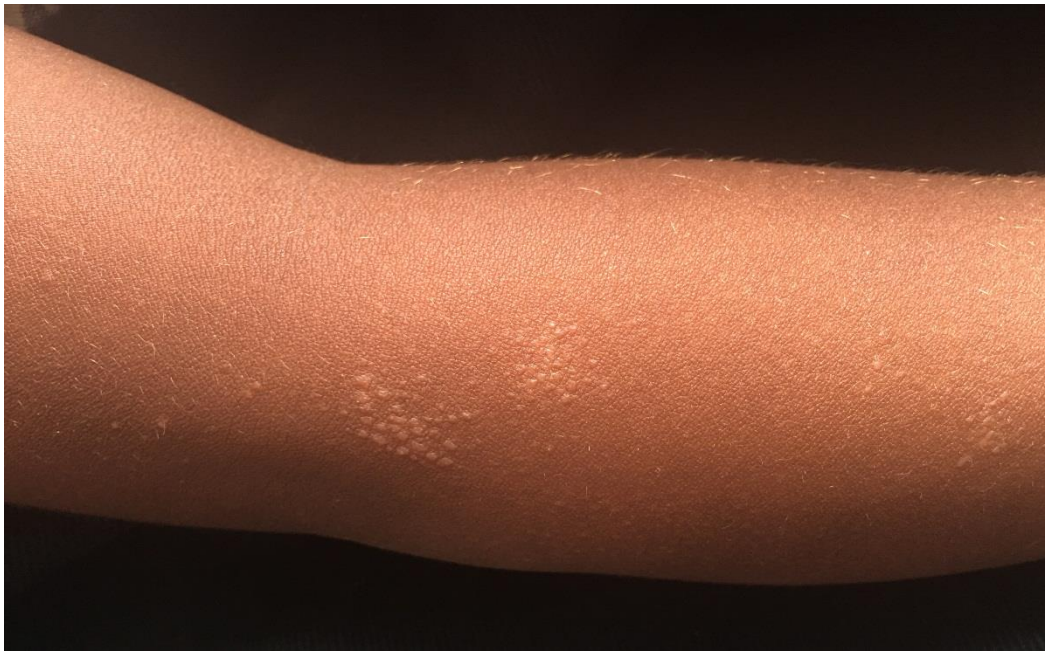

Figure 50: Multiple shiny skin coloured flat topped papules on right forearm

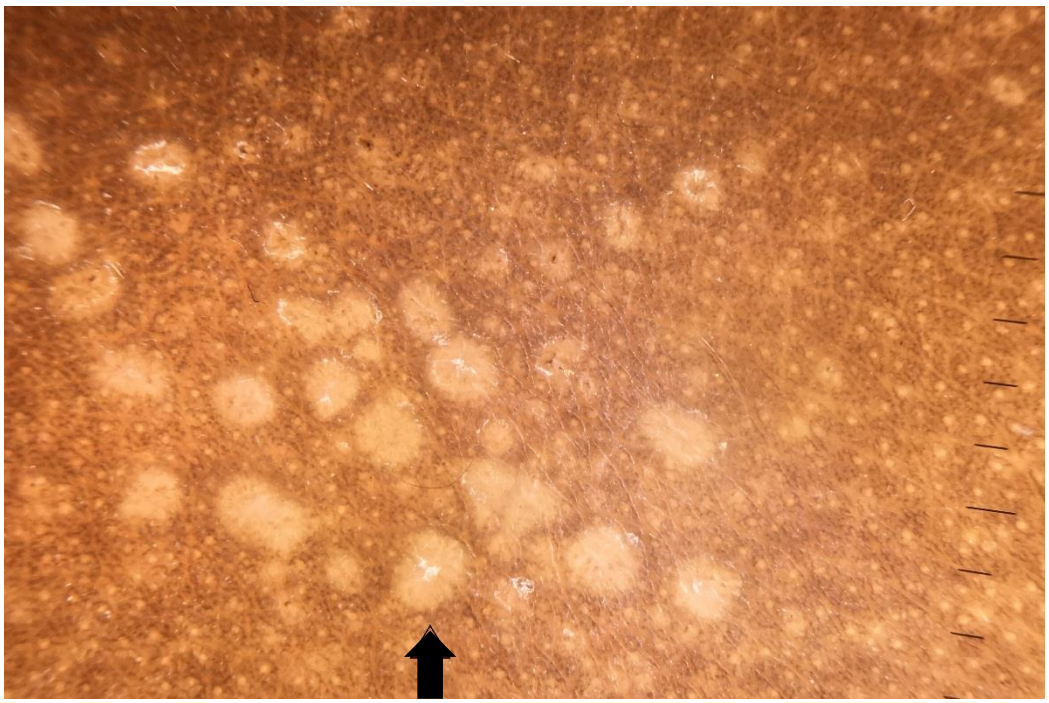

Figure 51: Lichen nitidus. On dermoscopy, white globules are seen

LICHEN STRIATUS

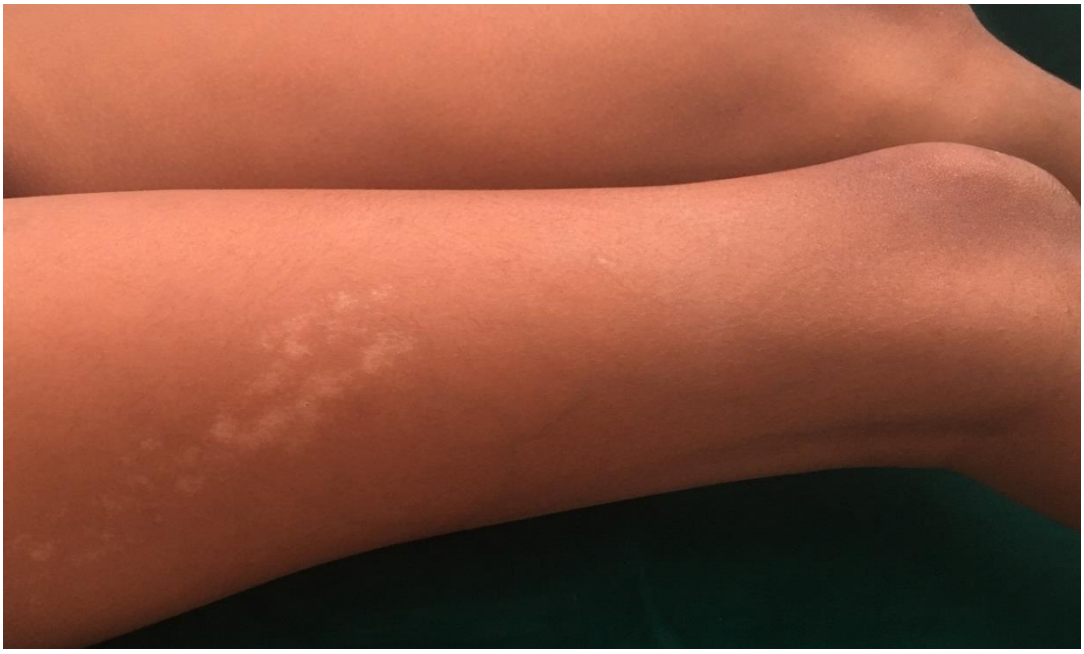

Figure 52: Hypopigmented grouped papules along blaschko's line over right thigh

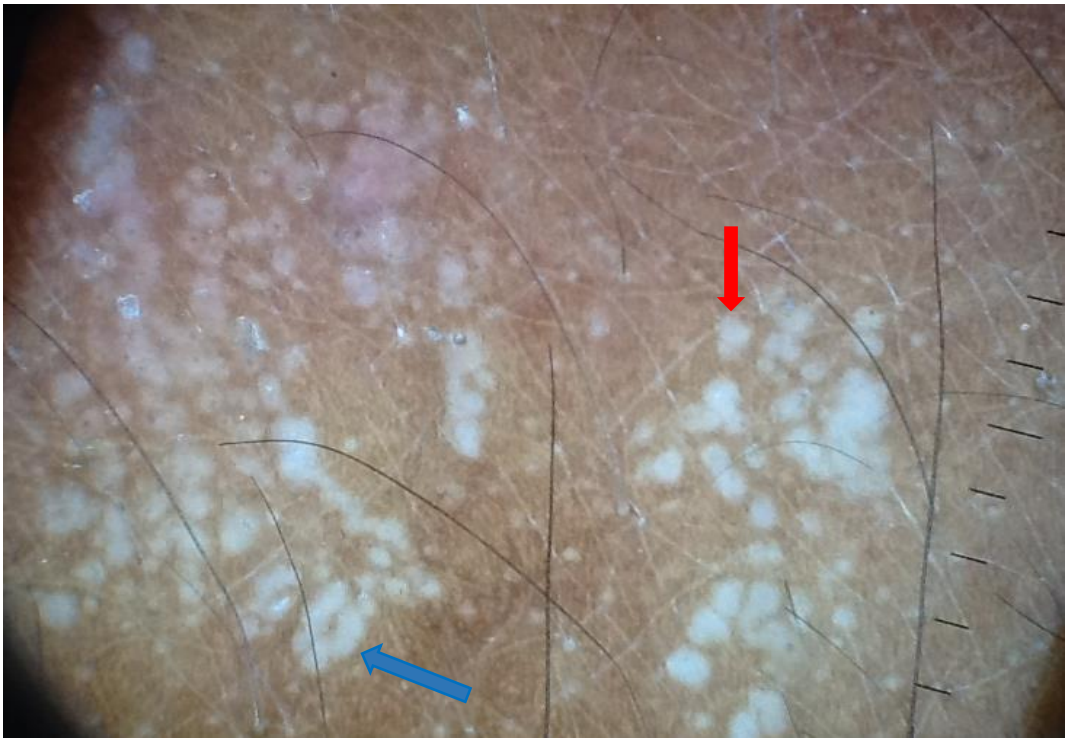

Figure 53: showing white globules (red arrow) coalescing to form cerebriform appearance (blue arrow).

DISCUSSION

DISCUSSION

Lichenoid dermatoses is a term used to describe various dermatological conditions which show interface dermatitis, on histopathology. The lichenoid dermatoses are Lichen planus and its variants, Lichenoid drug eruption, Lichen striatus, Lichen nitidus, Benign lichenoid keratosis, Ashy dermatosis, Graft versus host disease, Dermatomyositis and Lichenoid reaction in seborrheic keratosis (irritant type).[1]

Dermoscope is a recent, non-invasive diagnostic tool which helps in visualisation of very fine patterns of skin lesions and subsurface skin structures which are not visible to the naked eye.[2]

A total number of 100 lichenoid dermatoses cases, satisfying the inclusion criteria were screened and enrolled in the study. Out of which Lichen Planus was 45%, Lichen Planus hypertrophicus was 5%, Actinic Lichen Planus was 2%, Lichenoid drug eruption was 1%, Lichen planus pigmentosus was 5%, Lichen planopilaris was 7%, Lichen nitidus was 27%, Lichen striatus was 8%.

As lichenoid dermatoses can be present irrespective of the age of patients, similarly in this present study 0.9 year was the youngest subject and 84 year was the oldest subject. The mean age of patients in the present study was 29.34 years similar to another dermoscopic study done on 50 cases of lichenoid dermatoses which showed 38.14 years.[4]

Among the 100 cases, 51% were female and 49% were male in the present study where as 70% were female and 30% were male in a dermoscopic study done on 50 cases of lichenoid dermatoses.[4]

DERMOSCOPIC FEATURES OF LICHENOID DERMATOSES

LICHEN PLANUS

The common dermoscopic features of LP includes wickham's striae (WS), pigment patterns (grey-blue and brown dots), comedo, milium-like cysts and vascular structures. [1]

Wickham's striae is the hallmark finding in Lichen planus.[2] In our study, we noted wickham's striae (WS) in 91.1% (n=41) which is similar to an other study done in 25 patients with lichen planus where in WS was seen in 96%. However, 43 patients with lichen planus showed WS in just 37.2% in other study.[4]

WS disappears after treatment, suggesting that it can be used as an active marker in LP lesions.[39] WS may not be seen in few cases of lichen planus, this may be attributed to acute attack of the inflammatory cells and inadequate time to progress to hypergranulosis.

In Lichen Planus, on dermoscopy WS most commonly presents as white streaks in a reticular pattern and radial streaming and also circular, linear,

globular, veil-like, leaf venation, and starry sky/white dots are seen.[118] This can be due to distinct histopathological features in LP variants.[39]

Findings of morphology of WS in our study was in contrast to the other studies as mentioned in table in 10.[39, 117]

Table 10: The different morphologies of Wickham's striae in comparision with present study with that of other studies

Morphology of Wickham's striae	In our study (n= 45)	In a study done on 43 LP cases [117]	In a study done on 170 LP cases [39]
Radial streaming WS	63%	6.25%	5.2%
Reticulate WS	41.4%	25%	64.7 %
Veil like WS	17.07%	-	
Linear WS	14.6%	-	7.6%
Circular	7.3%	6.25%	1.1%
Reticular circular	4.8%	-	-
Leaf like	2.4%	37.5%	-

Colour of WS was observed as white in 82.9%, bluish white in 21.9% and yellow in 7.3% in present study similar to a study done in 170 patients where white colour was observed in 65.8%, blue white in 15.8% and yellow in 7.6%.[39]

WS is seen in active lesions and disappears with treatment, but pigment patterns resist treatment. Though all lesions appear at the same time, dermoscopic patterns differ according to location of lesion.[39]

The background colour of LP cases was violet in 75.5% in our study whereas in another study it was seen only in 38.8%.[39] The brown background was seen in 28.8% in our study similar to other studies which showed brown background in 17.6% and 24%.[39, 116] (Table 11)

Table 11: Showing background colour of LP lesions in present as well as other studies.

Background colour of LP	In our study (n= 45)	In a study done on 25 LP cases [116]	In a study done on 170 LP cases [39]
Violet	75.5%	-	38.8%
Brown	28.8%	24%	17.6%
Dull red	-	64%	-
Light red/pink	17.7%	12%	37.6%

Brown globules were seen in 60% cases in present study which is in contrast to an another study which showed brown globules in 20%.[18] (Table 12)

Table 12: Various pigment patterns of LP in comparision with present study and other studies

Pigment patterns of LP	In our study (n= 45)	In a study done on 15 LP cases[18]	In a study done on 43 LP cases[117]
Blackish blue globules	11.11 %	-	-
Greyish blue globules	17.7%	13.3%	-
Brown globules	60%	20%	-
Absent	20%	-	23.2%

The studies on dermoscopic findings on vascular patterns in Lichen Planus were similar to our study as mentioned in table 13.[18, 39, 117]

Table 13: Various vascular patterns of LP in comparision with present study and other studies

Vascular pattern of LP	In our study (n=45)	In a study done on 15 LP cases[18]	In a study done on 170 LPcases[39]	In a study done on 43 LP cases[117]
Red dots	11.1 %	20%	11.1%	18.6%
Red globules	40%	13.3%	4.7%	-
Red linear vessels	13.3%	-	11.1%	16.2 %
Peripheral homogenous	6.6%	-	-	-
Absent	46.6%	66.6%	72.9%	65.1 %

More evolved lesions show WS and vascular component. In long-standing lesions, grey-blue dotted areas, with or without WS and capillaries are seen.[115]

LICHEN PLANUS HYPERTROPHICUS

Greyish blue globules were seen in all 100% cases whereas pearly white areas with peripheral striations, brown globules, yellow structures and comedo like opening were seen in 80% cases and red globules were seen only in 20% cases (Table 14).

Table 14: Vascular and nonvascular features of lichen planus hypertrophicus in comparision with present study and other studies

Lichen planus hypertrophicus	In our study (n=5)	In a study done on 10 LPH cases[17]	In a study done on 24 LPH cases[42]
Pearly white areas with peripheral striations	80%	100%	100%
Grey blue globules	100%	60%	-
Brown globules	80%	-	54.6%
Red gobules	20%	60%	-
Yellow structures	80%	90%	66.6%
Comedo like openings	80%	30%	41.6%

ACTINIC LICHEN PLANUS

Dermoscopic findings of Actinic Lichen planus of other studies showed similar results to the present study as mentioned in table 15.[18, 39]

Table 15: Showing the percentage of vascular and nonvascular features of actinic lichen planus in comparision with present study and other

Actinic LP	In our study (n= 2)	In other study with 2 Actinic LP[18]	In other study with 10 Actinic LP[39]
Wickham's striae			
- present	-	-	-
- absent	100%	100%	100%
Background			
-Violet	50%	-	-
-Brown	50%	100%	100%
-Pink	100%	-	-
-No background	-	-	-
Pigment pattern			
-Blackish blue globules	-	-	-
-Greyish blue globules	-	-	-
-Brown globules	50%	100%	100%
-Absent	50%	-	-
Vascular pattern			
-Red dots	50%	-	-
-Red globules	-	-	-
-Red linear vessels	50%	-	-
-Peripheral homogenous	-	-	-
-Absent	-	100%	100%

LICHENOID DRUG ERUPTION

Only one case presented with lichenoid drug eruption secondary to Imatinib mesylate. On dermoscopy, wickham striae was absent with pink and brown background. Also observed were brown globules, red globules and comedo like openings.

LICHEN PLANUS PIGMENTOSUS

In our study, in all five cases of LPP diffusely arranged brown dots and globules in ‘peppering pattern’ with brown background was seen. In one case greyish blue background was seen along with brown background. The brown globules were seen predominantly around acrosyringeal and follicular openings. All these findings in the present study were in accordance to another study.[119] (Table 16)

The dotted pattern and circles result from aggregation of melanophages around eccrine and follicular involvement which is a distinguishing factor in Lichen planus pigmentosus.[119]

Table 16: Vascular and nonvascular features of lichen planus pigmentosus in comparision with present study and other studies.

Lichen planus pigmentosus	In our study (n= 5)	In other study with n=7 LPP cases[18]	In other study with n=46 LPP cases[84]
Wickham's striae			
- present	-	-	4.34%
- absent	100%	100%	95.6%
Background			
-Violet/ Greyish blue	20%	14.2%	-
-Brown	100%	28.5%	26.08%
-Pink	-	-	-
-Bluish black	-	28.5%	-
-No background	-	28.5%	-
Pigment pattern			
-Blackish blue globules	20%	-	8.6%
-Greyish blue globules	20%	57.1%	-
-Brown globules	100%	42.8%	-
-Absent	-	-	-
Vascular pattern			
-Red dots	-	-	-
-Red globules	-	-	-
-Red linear vessels	-	-	-
-Peripheral homogenous	-	-	-
-Absent	100%	100%	100%

The pigment pattern of LPP lesions in early phase show 'peppering' pattern which represents diffuse, singular melanophages in the superficial dermis. This may progresses to 'reticular' pattern which might be an incomplete and moderate form of the "perifollicular/annular" pattern.[39]

In some LPP lesions, the pigment pattern was absent in skin furrows, suggesting that the skin furrows are not exposed to friction, which could be the reason for the absence of pigmentation.[39]

LICHEN PLANOPILARIS

Out of seven cases of Lichen planopilaris screened, dermoscopic examination showed absence of wickham's striae in all cases, greyish blue globules seen in 71.4% cases as 'target pattern' of pigmentation, vascular pattern was absent in 85.7% similar to other studies.[18, 39] Perifollicular scales, cast and empty follicles were seen in 100% cases which is in accordance to other studies.[18, 39] (Table 17)

Table 17: Showing the percentage of vascular and nonvascular features of lichen planopilaris in our study and other studies

Lichen plano pilaris	In our study (n= 7)	In other study with n=15 LPp cases[39]	In other study with n=2 LPp cases[18]
Wickham's striae			
- present	-	40%	-
- absent	100%	60%	100%
Background			
-Violet/ Greyish blue	14.2%	33.3%	-
-Brown	42.8%	53.3%	100%
-Pink	14.2%	13.3%	-
-Bluish black	-	-	100%
-No background	28.5%	-	-
Pigment pattern	85.7%	86.6%	
-Blackish blue globules	-		8.6%
-Greyish blue globules	71.4%		-
-Brown globules	14.2%		-
-Absent	14.2%	13.4%	-
Vascular pattern			
-Red dots	-	20%	-
-Red globules	-	-	-
-Red linear vessels	-	-	-
-Peripheral homogenous	14.2%	-	-
-Absent	85.7%	80%	100%
Others			
-perifollicular scale/cast	100%	-	50%
-empty follicle	100%	-	100%
-structureless areas	57.14%	-	-

In Lichen planopilaris, the inflammatory phenomenon usually affects the hair follicles in a selective manner. In early stages, perifollicular scaling and casts with perifollicular erythema seen.[83]

In the fibrotic stage, whitish or milky-red areas are observed as a result of loss of follicular units.[12] Blue-violet areas and blue-grey dots reflects perifollicular pigment incontinence and appear as "target" pattern.[17]

The results show that different types of WS, pigment and vascular patterns can be seen according to the type of LP, location of lesion and duration of the disease.[39]

LICHEN NITIDUS

In lichen nitidus, we observed white globules in all 27 cases screened in the present study.

LICHEN STRIATUS

In our study, in all eight cases of Lichen striatus dermoscopy showed white globules coalescing to form cerebriform appearance which is similar to a case reported earlier.[103]

Wickham striae (WS) patterns and vascular patterns are not observed in LP actinicus, LPP and LPp lesions in early and active phase whereas pigment patterns can be seen which is consistent with the present study.[39] Different pigment patterns can be seen in the different lesions of the same patient at the same time and also different pigment patterns can be seen in the same lesions at different visits.[39]

CONCLUSION

CONCLUSION

- Dermoscopy helps in identifying fine structures which are not visible to the naked eye.
- Wickham's striae is the most common dermoscopic finding in Lichen Planus and other features like melanophages and blood vessels can also be visualized which helps in identifying the disease activity.
- Clinical use of dermoscopy in lichenoid dermatoses improves the diagnostic ability, can reduce the need for biopsy and to know the prognosis of the disease.

SUMMARY

SUMMARY

- Lichenoid dermatoses cases attending the Department of Dermatology at R.L Jalappa Hospital attached to Sri Devaraj Urs Medical College, Tamaka, Kolar from January 2017 to July 2018 were identified and a total of 100 cases satisfying the inclusion criteria were included in the study.
- A detailed history was taken, thorough examination was done, dermoscopy was performed and all the findings were documented as per proforma.
- Among the 100 cases, 51 were female and 49 were male.
- The youngest subject in the study was 0.9 year old and the oldest subject was 84 years old. The mean age of the study group was 29.34+/- 19.48.
- Among the 100 cases, Lichen Planus was 45% and its variants i.e Lichen Planus hypertrophicus was 5%, Actinic Lichen Planus was 2%. Other lichenoid dermatoses screened in the present study were Lichenoid drug eruption (1%), Lichen planus pigmentosus (5%), Lichen planopilaris (7%), Lichen nitidus (27%), Lichen striatus (8%).
- Patients with Lichen Planus (71.1%) and Lichen nitidus (74.1%) gave 0-3 months history of duration of lesions whereas patients with Lichen planus pigmentosus (40%) and Lichen planopilaris (42.8%) gave more than 12 months history of duration of illness.

- On examination, papules were commonly seen in Lichen planus (64.4%), Lichen nitidus (100%) and Lichen striatus (100%). Macules were seen in Lichen planus pigmentosus (100%) and alopecia in Lichen planopilaris (85.7%).
- Wickham's striae was seen only in lichen planus in 91.1% cases. The common morphological pattern of WS seen was radial streaming pattern (63%) and reticulate pattern (41.4%) with white colour (82.9%) wickham's striae being commonest.
- Brown globules was the commonest pigment pattern seen in Lichen planus (60%), Lichen planus hypertrophicus (80%) and Lichen planus pigmentosus(100%). Greyish blue globules were seen in Lichen planus hypertrophicus (100%), Lichen planopilaris (71.4%), Lichen planus (17.7%), Lichen planus pigmentosus (20%). Pigment pattern was absent in Lichen nitidus (100%) and Lichen striatus (100%).
- Diffuse type of pigmentation was seen in Lichen planus (100%), lichen planus hypertrophicus (100%) and Lichen planus pigmentosus (100%). Perifollicular type of pigmentation was seen in Lichen planus pigmentosus (100%) and Lichen planopilaris (71.4%).
- Red globules (40%) and peripheral homogenous erythema (6.6%) was seen in Lichen planus. No vascular patterns were seen in Lichen planus hypertrophicus (80%), Lichen planus pigmentosus (100%), Lichen planopilaris(85.7%), Lichen nitidus (100%) and Lichen striatus (100%).

- White globules were seen in Lichen nitidus (100%) and coalescing white globules were seen in Lichen striatus (100%).
- In Lichen planopilaris, empty follicles (100%), perifollicular scales and casts (100%) and structureless areas (57.1%) were seen.

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ANNEXURES

PROFORMA

Patient particulars **Case number**

NAME:	**OP/IP NUMBER**
AGE& GENDER:	**DATE:**
ADDRESS:	**Occupation:**

CHIEF COMPLAINTS:

Skin lesions- Itching / discoloration

Oral lesions- Discomfort/ burning sensation/ discoloration/ pain

Genitals lesions- Discomfort/ burning sensation/ discoloration/pain

HISTORY OF PRESENT ILLNESS:

Skin lesions: Onset of lesion, duration

Progression: slow/ rapid

Site: upper limbs(flexors / extensors), lower limbs(flexors / extensors), trunk.

Associated symptoms- Itching / discoloration / burning sensation on exposure to sunlight.

Oral lesions : Onset of lesion, duration

Progression: slow/ rapid

Site: buccal mucosa/ tongue/ lips/ gingiva/ hard palate/ soft palate.

Associated symptoms: Discomfort/ burning sensation/ discoloration/ pain/ ulcer.

Genitals lesions: Onset of lesion, duration

Progression: slow/ rapid

Site: glans, prepuce, coronal sulcus, shaft of penis

Associated symptoms: Discomfort/ burning sensation/ discoloration/pain.

Scalp: Hair loss (diffuse/ patchy)

Nail: nail changes, nail loss.

Systemic symptoms: Joint pains, pain abdomen, burning micturition.

History of drug intake: Drugs – Antihypertensive, Anti-tubercular, Anti-epileptics, NSAID's, Anti-malarials, Antibiotics, antifungals.

Risk factors:

- Smoking/ alcohol/betel and areca chewing/ other forms of tobacco chewing/ spicy foods.

- Stress
- Dentures

PAST HISTORY:

- Associated medical illness: Diabetes / TB/ HIV /Other skin lesions.
- Treatment history

PERSONAL HISTORY:

Food habits: Non vegetarian / vegetarian/ spicy diet

Bowel/ Bladder habits: regular/ altered.

FAMILY HISTORY:

Similar complaints:

Other skin problems:

<u>ON EXAMINATION:</u>

GENERAL PHYSICAL EXAMINATION:

Built and Nourishment:

Pallor/ Icterus/ Clubbing/ Cyanosis/ Significant lymph node enlargement/ Edema

Vitals: Temperature

Pulse

Blood pressure

Respiratory rate

CUTANEOUS EXAMINATION:

Morphology: clinical type of lesions, pattern of lesions, site of lesion, Secondary changes

Distribution of lesions

HAIR AND NAIL EXAMINATION

SYSTEMIC EXAMINATION:

- CVS
- RS
- PER ABDOMEN
- CNS

DERMOSCOPIC PATTERNS

1. Wickham's striae: Present/ Absent
2. Morphology of Wickham's striae:
    - Reticulate
    - Radial Streaming
    - Leaf Like
    - Linear
    - Veil Like
    - Circular
    - Circular Reticulate
3. Colour of Wickham's striae
    - BW- Bluish White
    - W- White
    - Y- Yellow
4. Back ground colour
    - V- Violet
    - B- Brown
    - P – Pink
5. Pigment pattern
    - BG- Brown globules
    - BBG- Blackish blue globules

- GBG- Greyish blue globules
- BD- Brown dots

6. Vascular pattern
    - RD- Red dots
    - RG- Red globules
    - RL- Red linear
    - PHE- Peripheral Homogenous Erythema
7. Additional findings
    - CLO- Comedo like openings
    - PWAPS- Pearly white areas with peripheral striations
    - YS- Yellow structures
    - IAO- Increased brown globules around acrosyringeal openings
    - PS- Perifollicular scales
    - PC- Perifollicular cast
    - EF- Empty follicle
    - SA- structureless areas
    - WG- White globules

INVESTIGATIONS:

1. Complete haemogram

2. Gram stain

3. KOH mount

4. Culture: Bacterial/Fungal

5. Biopsy: histopathology findings

6. Serology:

7. Others if any:

FINAL DIAGNOSIS:

TREATMENT:

PATIENT INFORMATION SHEET

Study title: DERMOSCOPIC PATTERNS IN LICHENOID DERMATOSES

Study site: R.L Jalappa Hospital ,Tamaka, Kolar.

Aim: To perform dermoscopy and document the findings in lichenoid dermatoses.

Purpose of this study is to analyse various dermoscopic patterns of lichenoid dermatoses. With the acceptance of standardized dermoscopic criteria worldwide as diagnostic test would eliminate the need for invasive histopathology. Thus, dermoscopy can help us to reduce the number of unnecessary invasive interventions.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in this study we will collect information (as per proforma) from you. Relevant blood investigations will be carried out if required. This information collected will be used for dissertation and publication only.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. The expenses required for the above investigations will be funded by the study investigator. This study has been reviewed by the Institutional Ethics Committee and you are

free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For any further clarification you can contact the study investigator:

Dr. Priya Prem

Mobile no: 9535490506

E-mail id: prempriya.b@gmail.com

CONSENT FORM

Study title: DERMOSCOPC PATTERNS IN LICHENOID DERMATOSES.

Chief researcher/ PG guide's name: DR. PRIYA PREM

Under the guidance of: DR. RAJASHEKAR T.S

Name of the subject:

Age :

Address :

a. I have been informed in my own vernacular language the purpose of the study, the necessity of relevant investigations to be carried out and photographs to be taken.
b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.
c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.
d. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

e. I confirm that _____ (chief researcher/ name of PG guide) has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that I may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

Participant's signature

Signature of the witness:

I have explained to _____ (subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Chief Researcher/ Guide signature Date:

KEY TO MASTER CHART

A. Serial number

B. Age

C. Gender

- M- Male
- F- Female

D. Symptoms

- IL- Itchy lesions
- NL- Non itchy lesions

E. Duration of illness

F. Drug history

G. Site

- UL- Upper limb
- LL- Lower limb
- T- Trunk
- F- Face
- P- Penis
- DH- Dorsum of hand
- N- Neck
- B- Back
- C- Chest

- S- Scalp
- F- Forehead

H. Signs

- VP- Violaceous papule
- VPq-Violaceous plaque
- VM- Violaceous macule
- P- Papule
- HP- Hypopigmented papule
- SGP- Slate grey pigmentation

I. Clinical diagnosis

- LP- Lichen Planus
- ZLP- Zosteriform Lichen Planus
- LLP- Linear Lichen Planus
- LPH- Lichen Planus Hypertrophicus
- LPP- Lichen planus pigmentosus
- LPp- Lichen Planopilaris
- LN- Lichen Nitidus
- LS- Lichen striatus
- LDE- Lichenoid Drug eruption
- AA- Alopecia Aerata
- DRIF- Disseminated and recurrent infunibulo-folliculitis

J. Wickham's striae morphology

- R- Reticulate
- RS- Radial Streaming
- LL- Leaf Like
- L- Linear
- VL- Veil Like
- C- Circular
- CR- Circular Reticulate

K. Colour

- BW- Bluish White
- W- White
- Y- Yellow

L. Background colour

- V- Violet
- B- Brown
- P - Pink

M. Pigment pattern

- BG- Brown globules
- BBG- Blackish blue globules
- GBG- Greyish blue globules
- BD- Brown dots

N. Vascular pattern

- RD- Red dots
- RG- Red globules
- RL- Red linear
- PHE- Peripheral Homogenous Erythema

O. Additional findings

- CLO- Comedo like openings
- PWAPS- Pearly white areas with peripheral striations
- YS- Yellow structures
- IAO- Increased brown globules around acrosyringeal openings
- PS- Perifollicular scales
- PC- Perifollicular cast
- EF- Empty follicle
- SA- structureless areas
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P. Final diagnosis

- LP- Lichen Planus
- LPH- Lichen Planus Hypertrophicus
- LPP- Lichen planus pigmentosus
- LPp- Lichen Planopilaris
- LN- Lichen Nitidus

- LS- Lichen striatus
- LDE- Lichenoid Drug eruption

serial number	Hospital number	Age	Gender	symptoms	Duration(in months)	drug history	site	signs	clinical diagnosis	ws morphology	WS colour	Back ground	Pigment pattern	vasculature pattern	additional features	Dermoscopic conclusion
1	366415	8	M	IL	1	-	T + UL + LL	VP	Eruptive LP	RS	w	-	-	RG	-	CLP
2	463770	38	F	IL	0.2	-	T + UL + LL	VP	LP	LL	BW	V	BBG	RG	-	CLP
3	479234	7	F	IL	0.5	-	T+ LL	VP + VPq	LP + LPH	RS, R	BW	V	BBG	RG	WG , CLO	LP + LPH
4	483355	45	M	IL	2	-	T + UL + LL	VP	LP	RS	w	-	-	RG, RL	-	CLP
5	374623	78	F	IL	6	-	Right LL	VP + VPq	ZLP	R, RS	BW	V	BBG	RD, RG	-	CLP
6	387834	41	F	IL	3	-	T+ LL	VPq	LP	R	w	V	-	-	-	CLP
7	476262	28	F	IL	2	-	Left LL	VP	ZLP	R	w	V, B	BG	RD, RG	WG	CLP
8	236291	30	M	IL	0.5	-	T + UL + LL	VP	LP	L	w	V	GBG & BG	RD	-	CLP
9	459950	37	F	IL	6	-	Left LL	VPq	ZLP	-	-	B	BG	-	-	CLP
10	294230	18	F	IL	6	-	Left LL	VPq	LLP	-	-	B	BG	-	-	CLP
11	459796	30	F	IL	1	-	T+ LL	VP + VPq	LP	RS	w	P	BG	-	-	CLP
12	414065	34	F	IL	4	-	T + UL + LL	VP + VPq	LP	R, RS, VL	w	B,P	BG	RG	-	LP
13	301842	30	M	IL	180	-	T+ LL	VP + VPq	LP + LPH	RS, LL	BW	V	BBG, BG	PHE, RG	CLO, PWASPS, YS	LP + LPH
14	493020	20	M	IL	1.3	-	T + UL + LL	VP	LP	L, RS	w	V	GBG	RD	-	LP
15	372760	26	F	IL	2	-	T + UL + LL	VP	LP	R	w	V	GBG	-	-	LP
16	319511	52	M	IL	0.8	-	T + UL + LL	VP	LP	R	w	V	GBG	-	-	LP
17	370499	35	F	IL	1	-	T + UL + LL	VP	LP	RS	w	-	-	RD	-	LP
18	533400	41	M	IL	12	-	T + UL + LL	VP	LP	RS	w	P,V	GBG	RG, RL	-	LP
19	522397	13	M	IL	6	-	UL + LL	VP	LP	RS	w	V	-	RG	-	LP
20	551595	35	F	IL	1	-	T + UL + LL	VP + VPq	LP	R, RS, VL, C, L, CR	w, BW	V, B	GBG & BG	RG, RL	-	LP
21	543189	45	F	IL	6	-	UL + LL	VP	LP	L,C	w	V	BG	PHE	-	LP
22	516340	28	M	IL	1	-	UL	VP	LP	RS	w	P	-	RG	-	LP
23	510709	15	F	IL	0.5	-	T + UL + LL	VP	LP	R, RS, VL	w, Y	V	BG	RG, RL	-	LP
24	438156	27	M	IL	5	-	LL	VP	LP	RS, VL	w, BW	V, B	BG	RG	-	LP
25	496492	56	F	IL	1	-	UL + LL	VP	LP	RS	w	V, B	-	-	-	LP
26	547381	35	F	IL	6	-	Right LL	VP	LLP	RS	w	B	-	-	-	LP
27	90805	46	M	IL	1	-	LL	VP	LP	R, RS	w, Y	V, B, P	BG	RG, RL	-	LP
28	501051	23	F	IL	1.5	-	T + UL + LL	VP	LP	RS	w	P	GBG, BG	RL	-	LP
29	559360	20	M	IL	3	-	Right LL	VP + VPq	ZLP	R	BW	V, B	BG	-	-	LP
30	570206	42	M	IL	0.2	-	UL + LL	VP + VPq	LP	R, RS, VL	w, BW	V, B	BG	RG	CLO	LP
31	426414	23	F	IL	0.3	-	F	VP	LP	VL, L	w	V	BG	-	-	LP
32	580705	25	F	IL	1	-	UL + LL	VP	LP	L	w	V, B	BG	-	-	LP
33	551225	52	M	IL	3	-	UL + LL	VP	LP	R, RS, L	w	P,V	BG	RG	CLO	LP
34	580706	54	F	IL	1	-	T+ LL	VP	LP	VL, CR	w	V	BG	PHE, RG	-	LP
35	364753	38	M	IL	1.5	-	UL + LL	VP	LP	RS	w	P,V	BG	-	-	LP
36	297356	60	M	IL	24	-	UL + LL	VP	LP	RS	w	V	BG	-	-	LP
37	599048	22	M	IL	2	-	UL	VP	LP	RS	w	V	BG	-	-	LP
38	594524	50	M	IL	1	-	UL + LL	VP	LP	RS	w	V	GBG, BG	-	CLO	LP
39	608009	50	M	IL	1	-	T + UL	VP	LP	C	w	V	-	-	-	LP
40	377964	32	M	IL	2	-	P	annular VPq	Annular LP	-	-	V	-	-	-	LP
41	623175	55	F	IL	3	-	LL	VP	LP	-	-	V	BG	-	-	LP
42	269362	44	F	IL	6	-	UL + LL	VP + VPq	LP	R	BW	V	BBG	-	-	LP
43	317760	42	M	IL	2	-	Left LL	VP + VPq	ZLP	R	w	V	BG	-	-	LP
44	412223	65	M	IL	4	-	UL + LL	VP + VPq	LP	R	Y	V	BG	-	-	LP
45	305233	44	M	IL	2	-	UL + LL	VP + VPq	LLP	RS	w	B	BG	-	-	LP
46	352993	67	M	IL	1	-	LL	VPq	LPH	-	-	-	BG, GBG	-	PWAPS, CLO, YS	LPH
47	342302	72	F	IL	12	-	UL + LL	VPq	LPH	-	-	-	BG, GBG	-	PWAPS, YS	LPH
48	404884	84	M	IL	3	-	LL	VPq	LPH	-	-	-	BG, GBG	-	PWAPS, CLO, YS	LPH
49	301842	30	M	IL	180	-	T+ LL	VP + VPq	LP + LPH	RS, LL	BW	V	GBG, BG	PHE, RG	CLO, PWASPS, YS	LP + LPH

50	479234	7	F	IL	0.5	-	T+ LL	VP + VPq	LP + LPH	RS, R	BW	V	GBG	RG	WG , CLO	LP + LPH
51	474195	5	F	NL	3	-	T + UL + LL	P	LN	-	-	-	-	-	WG	LN
52	358293	6	M	NL	3	-	UL	P	LN	-	-	-	-	-	WG	LN
53	464648	5	F	NL	2	-	DH	P	LN	-	-	-	-	-	WG	LN
54	459242	34	M	NL	1	-	DH	P	LN	-	-	-	-	-	WG	LN
55	487745	4	F	NL	0.5	-	DH	P	LN	-	-	-	-	-	WG	LN
56	383135	5	F	NL	6	-	UL	P	LN	-	-	-	-	-	WG	LN
57	150449	5	M	NL	1	-	Right UL + LL	P	LN	-	-	-	-	-	WG	LN
58	491399	9	M	NL	2		T + UL + LL	P	LN	-	-	-	-	-	WG	LN
59	368190	4	F	NL	1	-	UL	P	LN	-	-	-	-	-	WG	LN
60	463154	27	M	NL	0.3	-	T+ LL	P	LN	-	-	-	-	-	WG	LN
61	360689	12	F	NL	0.2	-	UL	P	LN	-	-	-	-	-	WG	LN
62	337007	32	M	NL	2.5	-	LL	P	LN	-	-	-	-	-	WG	LN
63	358296	15	F	NL	4	-	UL	P	LN	-	-	-	-	-	WG	LN
64	348496	18	M	NL	1	-	UL	P	LN	-	-	-	-	-	WG	LN
65	507781	4	M	NL	0.6	-	T + UL + LL	P	LN	-	-	-	-	-	WG	LN
66	507214	0.9	F	NL	0.1	-	T + UL + LL	P	LN	-	-	-	-	-	WG	LN
67	547435	36	M	NL	1	-	UL	P	LN	-	-	-	-	-	WG	LN
68	560707	20	F	NL	1	-	UL	P	LN	-	-	-	-	-	WG	LN
69	579426	15	F	NL	4	-	F, UL	P	LN	-	-	-	-	-	WG	LN
70	581586	9	F	NL	1	-	F, N	P	LN	-	-	-	-	-	WG	LN
71	571544	9	M	NL	12	-	F, UL	P	LN	-	-	-	-	-	WG	LN
72	566029	3.5	F	NL	1	-	UL, LL	P	LN	-	-	-	-	-	WG	LN
73	581264	37	F	NL	12	-	UL, LL	P	LN	-	-	-	-	-	WG	LN
74	585156	2.5	F	NL	0.2	-	UL	P	LN	-	-	-	-	-	WG	LN
75	398674	35	M	NL	5	-	UL, LL	P	LN	-	-	-	-	-	WG	LN
76	566572	25	F	NL	2	-	UL	P	LN	-	-	-	-	-	WG	LN
77	628414	36	F	NL	12	-	UL	P	LN	-	-	-	-	-	WG	LN
78	442936	20	F	D	6	-	F+ T + UL +LL	SGP	LPP	-	-	B	diffuseBD	-	IAO	LPP
79	86550	14	F	D	0.6	-	F	VM	LPP	-	-	B	diffuse BD, BBG	-	IAO	LPP
80	166781	46	F	D	0.8	-	F + N	VM	LPP	-	-	V,B	diffuse BD, GBG	-	IAO	LPP
81	495492	9	M	D	0.5	-	F+ UL	SGP	LPP	-	-	B	diffuse BD	-	IAO	LPP
82	538081	40	F	D	60	-	F+ UL	SGP	LPP	-	-	B	diffuse BD	-	IAO	LPP
83	511639	0.9	M	NL	3	-	Left UL, B	HP	LS	-	-	-	-	-	coalesing WG	LS
84	511798	10	F	NL	24	-	right LL	HP	LS	-	-	-	-	-	coalesing WG	LS
85	472283	13	F	NL	6	-	right UL	HP	LS	-	-	-	-	-	coalesing WG	LS
86	341622	45	M	NL	6	-	right UL	HP	LS	-	-	-	-	-	coalesing WG	LS
87	365639	3	M	NL	12	-	right cheek	HP	LS	-	-	-	-	-	coalesing WG	LS
88	353532	12	M	NL	3	-	right LL	HP	LS	-	-	-	-	-	coalesing WG	LS
89	571569	6	F	NL	1	-	left UL	HP	LS	-	-	-	-	-	coalesing WG	LS
90	570244	7	M	NL	12	-	left UL	HP	LS	-	-	-	-	-	coalesing WG	LS
91	558021	30	M	LOH	60	-	S, LL	LOH	AA	-	-	P	-	PHE	PF,PC, EF, SA	LPp
92	568860	43	M	IL	240	-	T	P	DRIF/ LPp	-	-	-	GBG	-	PF, EF	LPp
93	479363	45	F	LOH	120	-	LL	LOH, M	LPp	-	-	B	GBG	-	PF, EF	LPp
94	621285	11	F	LOH	8	-	S	LOH	LPp	-	-	V	GBG	-	PF, PC, EF, SA	LPp
95	620766	25	M	LOH	2	-	S	LOH	LPp	-	-	B	GBG	-	PF, EF	LPp
96	598591	54	M	LOH	6	-	S	LOH, M	LPp	-	-	B	GBG	-	PF, PC, EF, SA	LPp
97	596873	58	F	LOH	12	-	T	LOH, M	LPp	-	-	-	BG	-	PF, EF	LPp
98	443101	72	M	IL	2	Imatinib x 7m	T + UL + LL	VM	LDE	-	-	B, P	BD & BG	RG	CLO	LDE
99	473242	35	F	IL + PS	0.2	-	C	VP	Actinic LP	-	-	B, P	-	RL	-	Actinic LP
100	449225	23	M	IL + PS	12	-	FH	VPq	Actinic LP	-	-	V & P	BG	RD	-	Actinic LP